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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

EDITORS
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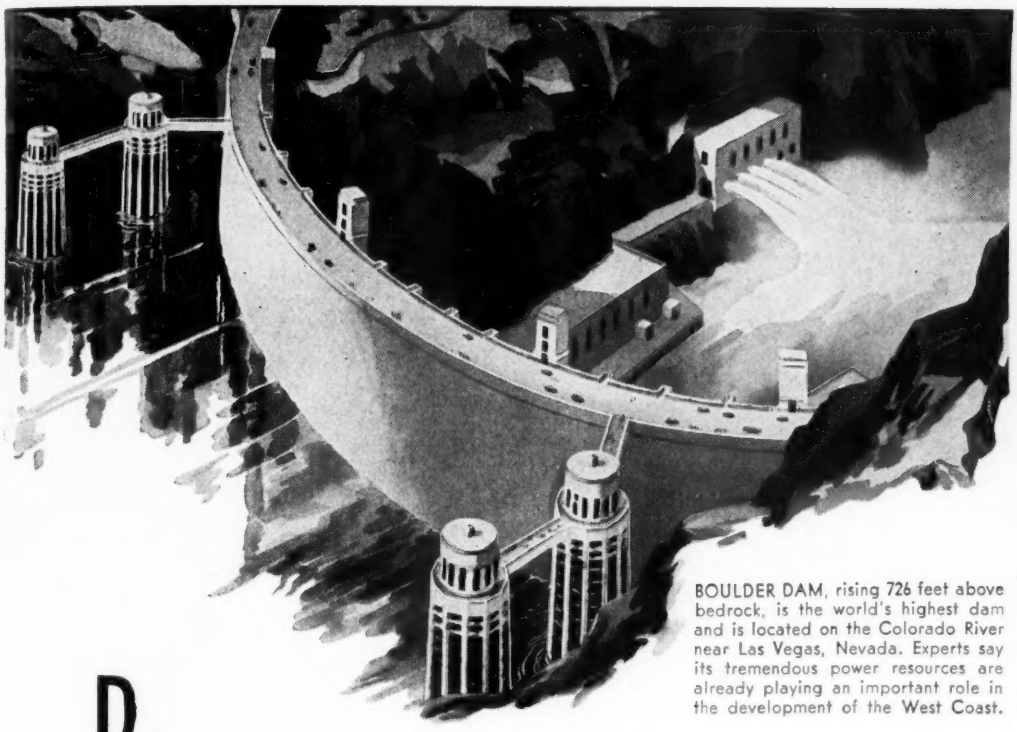

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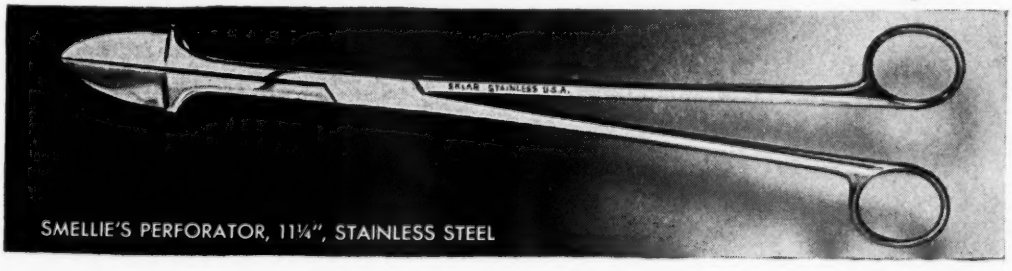

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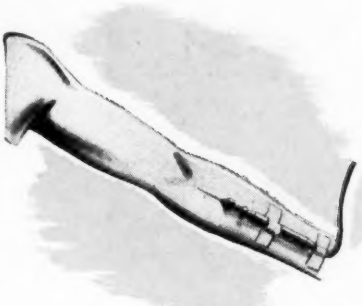

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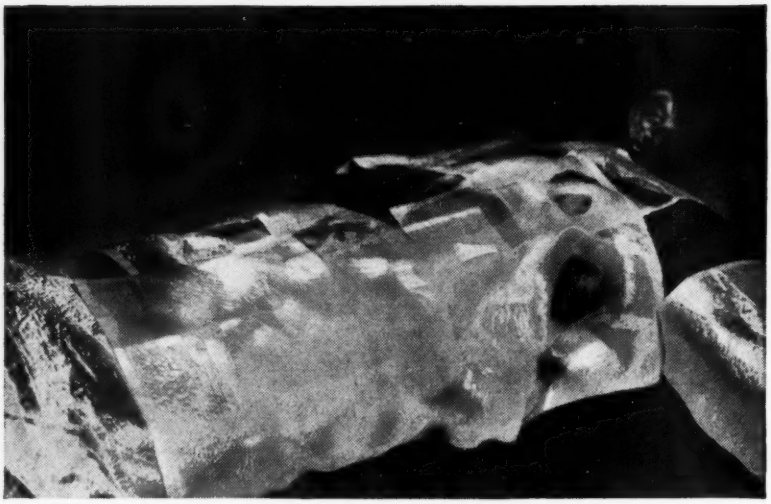

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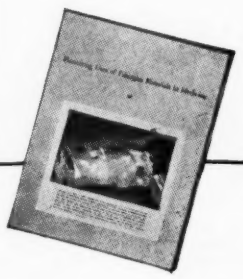

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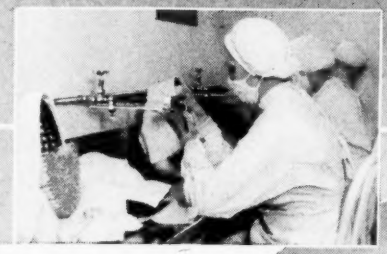

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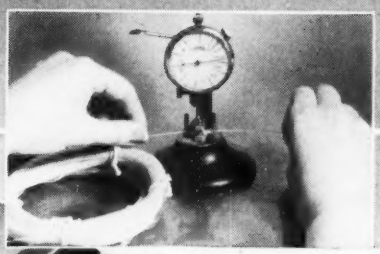

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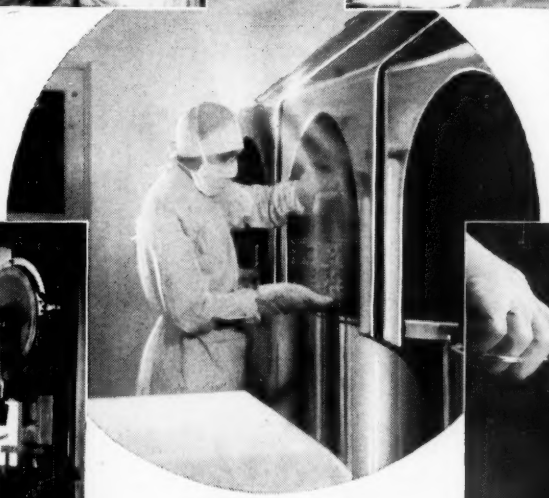

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(1). Spies, Tom D.; Cogswell, Robert C., and Vilter, Carl: J.A.M.A. (Nov 18) 1944. Spies, Tom D.: Med. Clin. N. Am. 27:273, 1943. (2). Jolliffe, Norman, and Smith, James J.: Med. Clin. N. Am. 27:567 (March) 1943.

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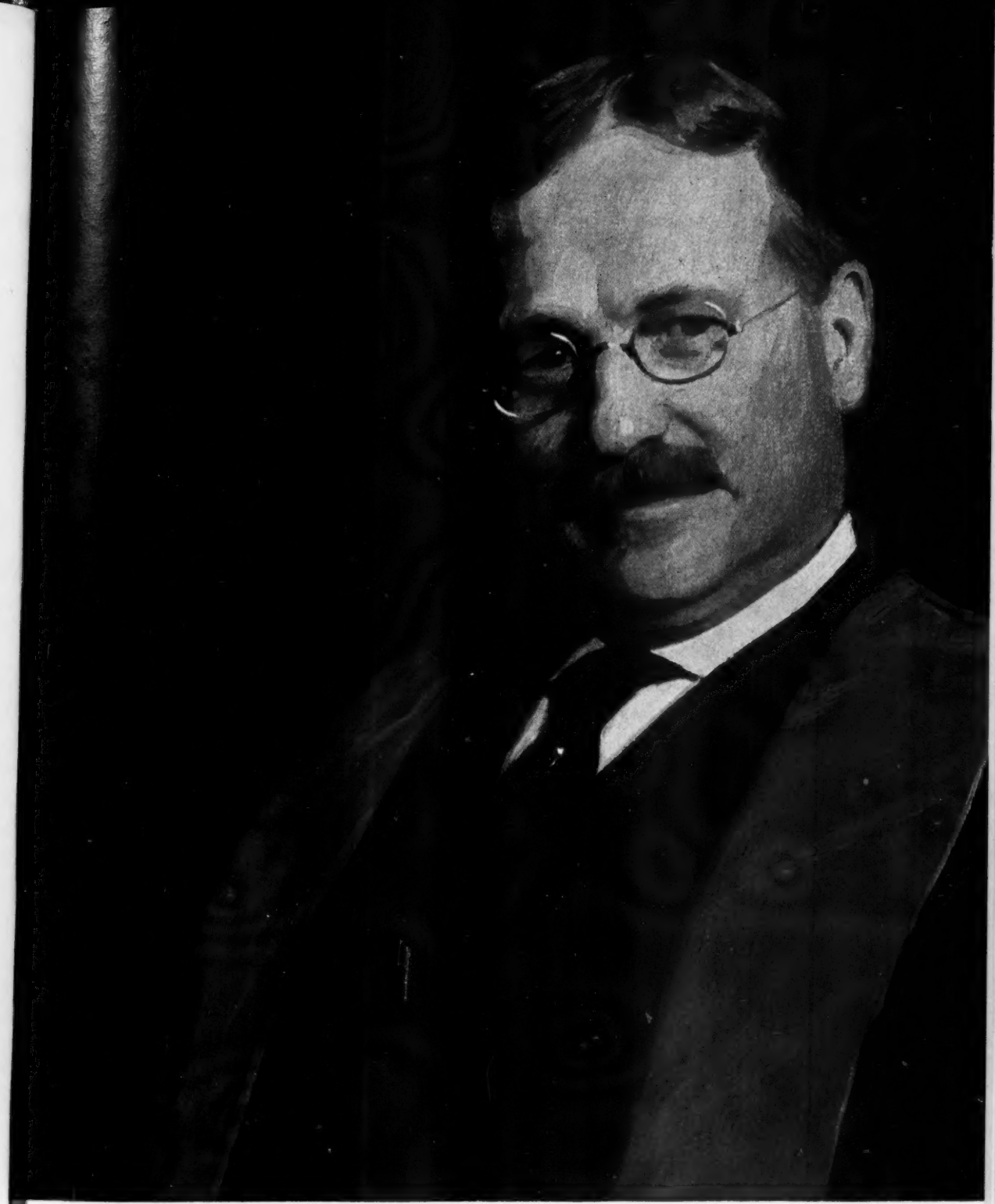

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John M. T. Finney

1863-1942

Master Surgeon, Author and Educator. Co-founder and first President, American College of Surgeons, 1913-1916. Brigadier General and Chief Consultant in Surgery, American Expeditionary Forces, 1917-1918. Staff member, later acting head, Department of Surgery and Surgeon-in-Chief, Johns Hopkins University, School of Medicine and Hospital, 1889-1925. Life trustee, Princeton University.

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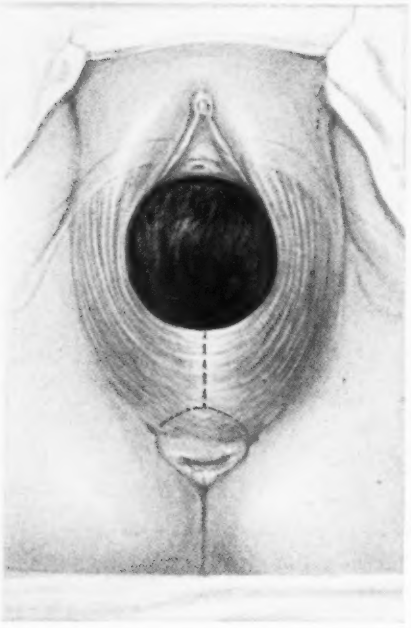
ETHICON SUTURE LABORATORIES, DIVISION OF JOHNSON & JOHNSON, NEW BRUNSWICK, N. J.

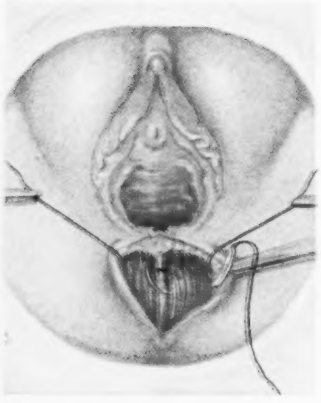
Median Episiotomy and Repair

(MATSNER)

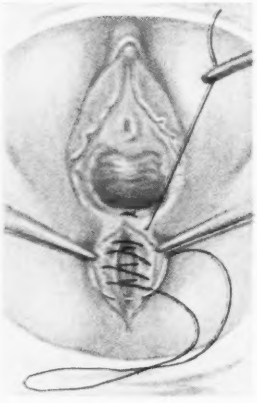


1. Vertical line indicates normal extent of incision. Arc line indicates elective extension, avoiding anal sphincters.

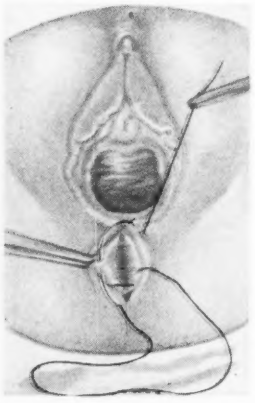
2. Incision is made in avascular midline area, separating the levator ani muscles. Tip-guard scissors used to avoid fetal trauma.



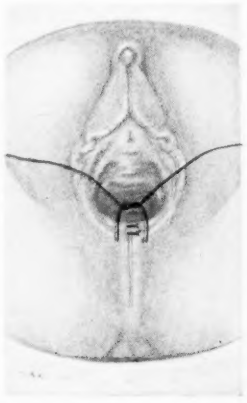
3. Vaginal mucous membrane sutured with interrupted chromic 00. Levator ani muscles approximated with interrupted chromic 0.



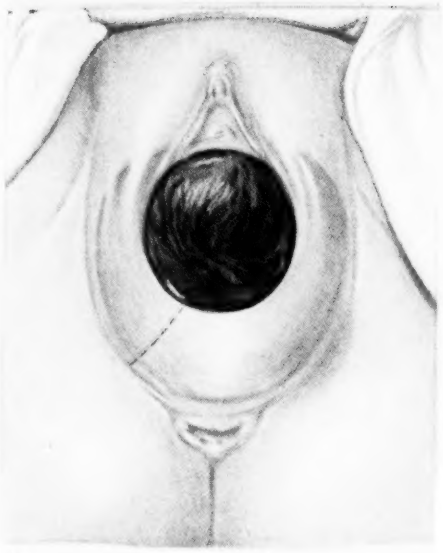
4. Continuous subcutaneous suture, chromic 00, beginning at upper angle and extending full length of incision.



5. Same suture used for subcuticular closure beginning at base of incision and ending opposite primary introduction.



6. Subcutaneous subcuticular repair completed. Note plastic result and absence of irritating external sutures.



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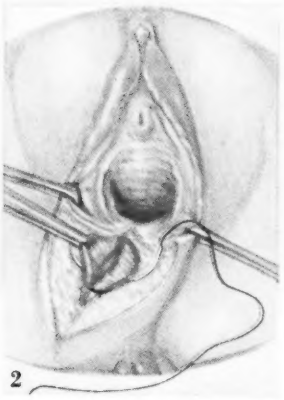

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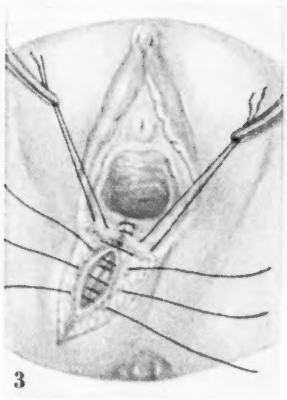

3

Mediolateral Episiotomy and Repair

1. Dotted line indicates usual extent and direction of incision which avoids possible extension into sphincter area. Incision can be made on either side.

2. Vaginal mucous membrane sutured with interrupted chromic 00. Deep interrupted chromic 0 sutures are used to approximate muscle.

3. Interrupted chromic 00 at mucocutaneous junction held by clamps until subcutaneous sutures are completed.

4. Skin closure completed by interrupted chromic 00. Note all suture ends are cut short to minimize irritation.

4

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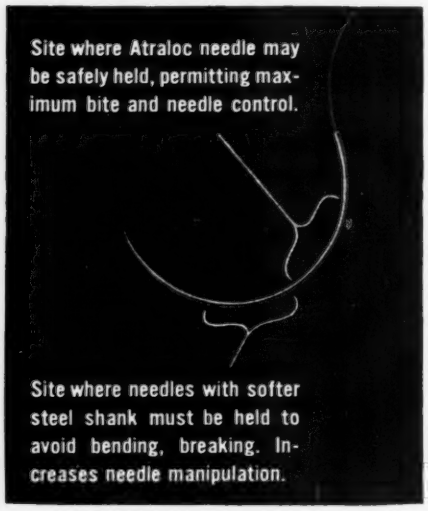
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Site where needles with softer steel shank must be held to avoid bending, breaking. Increases needle manipulation.

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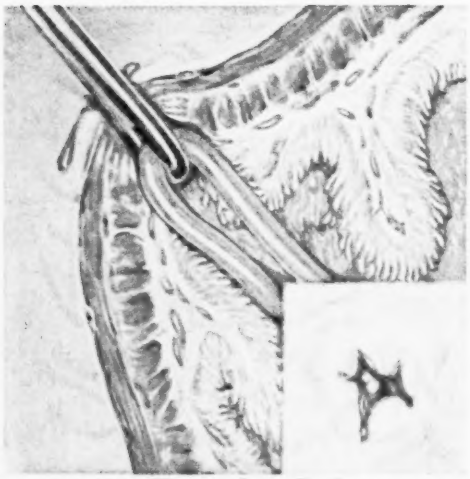
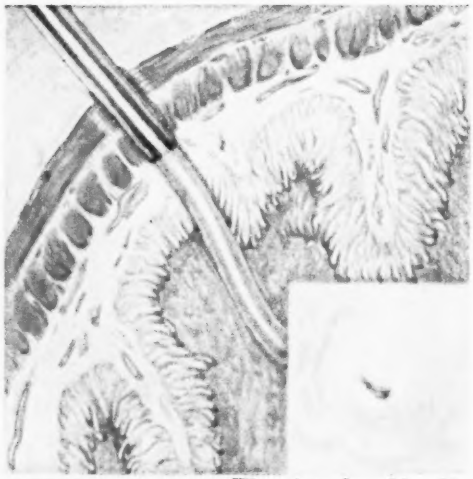


EYED NEEDLE: The eyed needle does greater injury to tissue because it pulls a looped suture in its wake.



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SURGERY

VOL. 17 APRIL, 1945 NO. 4

Original Communications

ACUTE PANCREATITIS

JOHN MORTON, M.D., ROCHESTER, N. Y.

(From Department of Surgery, School of Medicine and Dentistry and Strong Memorial Hospital)

PREVIOUS to 1938 the diagnosis of acute pancreatitis was, at best, uncertain. It was a diagnosis made from the clinical picture of an acute abdominal emergency, often alarming in its intensity, and supposedly calling for urgent surgery. The diagnosis was likely if severe epigastric pain struck suddenly in a middle-aged obese individual. If this was accompanied by vomiting, shock, tenderness in the epigastrium verging toward the left, absence of fever, a relatively rapid pulse, and a high white blood count, the diagnosis became more certain. Transverse radiation of pain across the upper abdomen, increasing distention, and tenderness in the left costovertebral angle would give additional support. Attempts would be made to rule out perforated peptic ulcer by x-ray examination; acute biliary tract disease, jaundice often being present; intestinal obstruction or mesenteric thrombosis; coronary occlusion by electrocardiographic examination; and peritonitis. When everything had been done, the attendants would be oftentimes in a dilemma. Operation would be advised to resolve the anxiety and to avoid missing an acute surgical condition.

What was needed at this time was a dependable, easily performed laboratory test. This test would of necessity be one which could be done without elaborate apparatus as it would have to be done at any time, day or night. And it would have to be a test which could be finished quickly. Somogyi[31a] gave us such a test. Other tests which had been in use, such as the Wohlgemuth test (starch splitting), did not permit measurement of small changes and were too time consuming. The viscosity of starch paste under action of diastase was variable and inadequate (Somogyi[31b] and Polowe[25]). The serum lipase test required a twenty-four-hour wait and was elevated in pancreatic cancer as well

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as in pancreatitis (Johnson and Bockus). Polowe's cuprous oxide precipitation demanded thirty minutes' incubation, then centrifuging, etc.

Somogyi's rapid method (amyloclastic activity) is a quantitative measurement of the *reaction time* and not of the reducing power of the reaction products. The results obtained by this procedure are computable in terms of reducing power and identical with such reductions. This permits estimation of low values and accuracy in high ranges (Somogyi[31a]). The test can be reported in twenty minutes. The blood amylase content in healthy persons is maintained at fairly constant levels. Eighty per cent of healthy ambulatory adults have levels between 80 and 150; the remainder have a low range of 60 to 80, or a high range of 150 to 180. Blood amylase values below 60 and above 200 are considered abnormal. The origin of the diastase content in the normal blood is at present unknown. It is completely absent in the newborn infant; appears at 2 months, is measurable at 3 months, and reaches normal at 1 year. It is not influenced afterward by age, sex, amount or type of food, fasting, dehydration, diuresis, exercise, or sleep. Under normal conditions neither the pancreas, salivary glands, nor liver contributes to the blood diastase level. Acute pathologic changes in the pancreas or salivary glands may cause a temporary effusion from these glands into the blood stream. This causes an increase in the diastase (amylase) content of the blood. The increase takes place quickly, reaches a high level and gradually subsides to normal within a few days.[21-23b] Injury to the pancreas tends to increase the amylase activity; injury to the kidney or to the liver decreases it (Polowe[25]). The serum amylase can be useful in differentiating perforated peptic ulcer from pancreatitis and also in determining the site of perforation according to Probstein and his co-workers.[27] When the serum amylase reading is low or subnormal in sharp epigastric pain it rules out acute pancreatitis or perforating ulcer into the pancreas; very high readings indicate acute pancreatitis; moderate elevations may mean perforation into the pancreas. Aspirated fluid from the peritoneal cavity can be tested for amylase activity in a doubtful case. In acute pancreatitis the peritoneal exudate gives high readings.

Etiology.—The fundamental cause for the behavior of the pancreatic ferments in pancreatitis still remains in debate. There is no question that living tissue is not affected by the pancreatic enzymes. Whipple and Goodpasture[32] demonstrated that the pancreatic juice was harmless intraperitoneally and intravenously. Biopsy and operations on the pancreas are not followed by pancreatic necrosis. When bile is injected into the pancreatic duct, pancreatic necrosis may occur. This is because of the local cytolytic and destructive properties of bile salts and not because of activation of intraductal trypsinogen. Active trypsin is incapable of digesting living tissue; it acts as a catalyst in facilitating the hydrolysis of proteins by the alkali of the pancreatic juice. The locally

destructive action of pancreatic juice is dependent on the concentration of the alkali. Infection of the injected bile augments the destructive action. The toxic effect is neutralized by proteins of the blood serum. Hemorrhage is probably a protective mechanism (Gaither[12]). Jones[18] does not agree with this. The pancreatic ducts which are not obstructed remain unharmed but anything which partially or completely blocks the ducts may cause acute pancreatitis. There are five principal contributing causes: (1) trauma, accidental or surgical, (2) infections with extension to the pancreas, biliary tract, duodenal ulcer, etc., (3) toxic, alcohol, drugs (arsphenamine), anesthesia, (4) obstructions to biliary tract, stone, spasm, infection, tumor, duodenal diverticulum, Ascaris, and (5) circulatory factors, stasis, hypertensive apoplexy, thrombosis, and embolism.

Dragstedt and his associates claimed that 60 per cent of acute pancreatitis arises in patients with antecedent biliary tract disease. In our series the figure was 62 per cent. In 10 per cent of these cases there is the common channel of Opie. The rest of the cases have spasm of the sphincter of Oddi as described by Archibald (Jones[18]). Lynch[20] found duct obstruction the main etiologic event, although vascular occlusion and infection caused a considerable proportion of the acute disease. Gaskell[13] claims that there is a form of acute necrosis of the pancreas as a cause of sudden death. Trauma of the head of the pancreas can produce this result. He cites ten cases with post-mortem examinations to support his views. Smyth[30] believes the vascular factors to be important and demonstrated all the pictures of acute pancreatitis following mercury injections into the arterial supply of the pancreas of dogs. Ackerman[1] reported a fatal case of acute hemorrhagic pancreatitis following transfusion of mismatched blood. Severe instances of acute disease following heavy drinking bouts have been noted by several authors. We have seen a number of such instances. Ogilvie[24] reported three cases of pancreatic necrosis associated with duodenal diverticula, thus favoring an obstructive etiology. Allergy has been implicated by Brodie and Ficarra.[3]

Pathology.—There are at least two types of acute pancreatitis. The more common form is acute pancreatic edema, called by Elman interstitial pancreatitis. This manifests itself by a boggy swollen pancreas with fluid under tension and is accompanied by fat necrosis in a large proportion of cases. The disease does not progress to hemorrhage and necrosis but subsides spontaneously in most instances if not disturbed.

The other type is hemorrhagic necrosis. This is a serious disease with death of a large part of the organ.

Sequelae.—As sequelae to these forms we may have acute pancreatic abscess, an immediate or late result of autolyzed infected necrotic tissue, or pseudocyst of the pancreas where the above necrotic tissue is walled off and the infection has burned out.

The acute edematous pancreatitis is followed by chronic inflammation in a large percentage of cases. When the disease has subsided and the patient is explored later, a hard enlarged pancreas is frequently encountered. Experimentally, Ireneus[16] has showed that the acinar cells tend to recover and become functionally efficient in about four weeks in experimental animals. Residual changes consist of interlobar and interacinar fibrosis, lymphocytic infiltration, and edema.

Glycosuria is noted in about 11 per cent of acute pancreatitis cases. Shumacker[29] claims that at least 2 per cent of all patients with severe acute pancreatitis develop diabetes; and of those surviving 3 to 10 per cent will develop it in time. Systematic follow-up studies should be made.

Diagnosis.—The symptoms are too well known to demand comment. Suffice it to say that acute pancreatitis can be diagnosed only rarely without the special amylase test, nor can the acute edematous form be differentiated from pancreatic necrosis at the beginning of the attack. Severe pain, vomiting, constipation, distention, and tenderness are common to many conditions in the abdomen and chest. Even the electrocardiogram may show changes suggestive of myocardial infarction or coronary thrombosis. The triad of upper abdominal peritoneal irritation, elevated serum amylase test, and electrocardiographic changes should establish the diagnosis of acute pancreatitis (Gottesman and coworkers.[14]). Perforated peptic ulcer, coronary disease, tabetic crises, peritonitis, severe biliary tract disease, acute intestinal obstruction, mesenteric thrombosis, and ectopic pregnancy must all be considered in differential diagnosis. By the course in the hospital it should be possible to separate the pancreatic edema from pancreatic necrosis. The former usually gives history of previous attacks, less prostration at the onset, and a high amylase reading. Over the course of three to five days the attack subsides spontaneously with return to normal. Pancreatic necrosis on the other hand is almost always prostrating at the start; the amylase test may or may not be elevated; the patient fails to improve during the conservative treatment (Casberg[6] and Elman[10b]); and if nothing is done, he dies or goes through a prolonged illness with sequelae such as abscess or cyst formation.

There may be repeated attacks of acute pancreatitis. Four of our patients had secondary attacks while in the hospital after the first attack had subsided. Two had second acute attacks within one year, and one patient had three separate acute attacks within a period of two years.

Treatment.—In acute edematous pancreatitis, diagnosed by amylase test, the curve should be followed and treatment should be conservative. Immediate operation is contraindicated. After the acute disease has subsided, the gall bladder should be studied and interval operation

carried out. The passage of small gallstones is frequently the cause of an acute attack of pancreatitis. In one of our patients a small stone was vomited during the height of a pancreatitis attack with prompt amelioration and recovery. Certainly in any of these patients with biliary tract disease there is a rationale for exploring the common duct and dilating the lower end to abolish sphincter spasm. Some surgeons

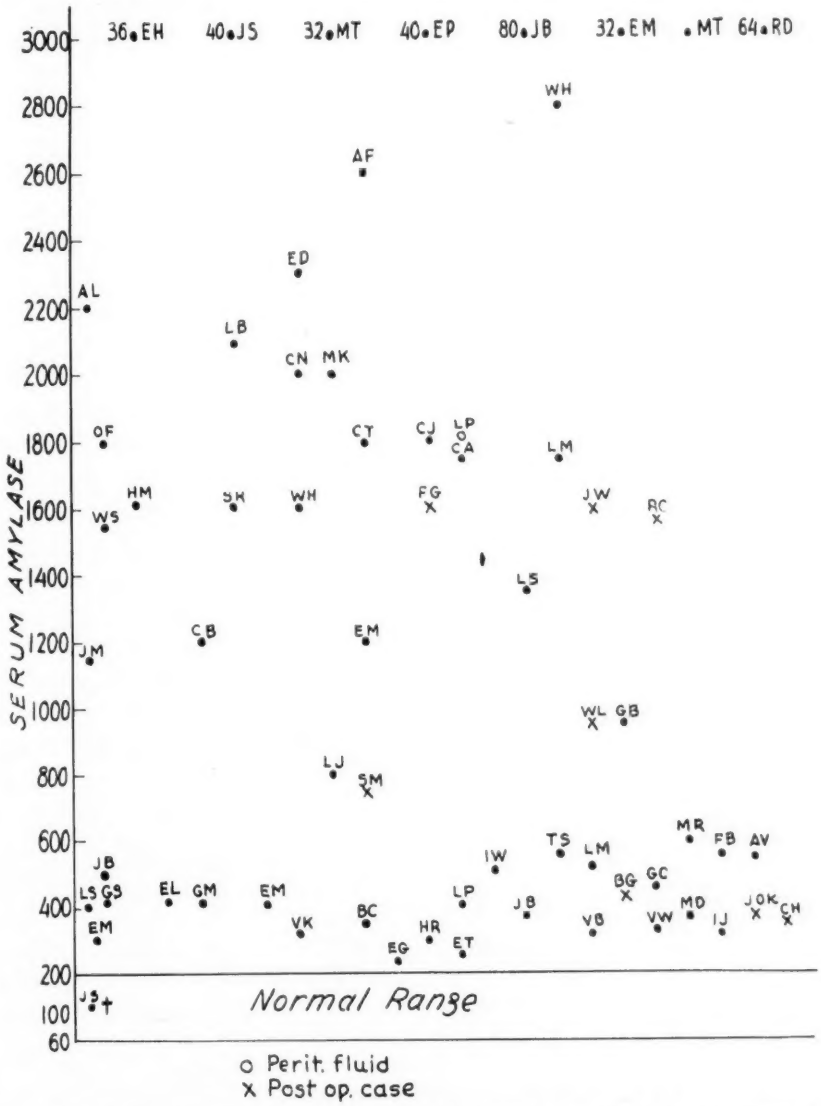

Fig. 1.—Acute pancreatitis gives serum amylase above the normal range except late in the onset and after the pancreas is necrotic.

advocate drainage of the common duct for three to four weeks but this, to my mind, would depend on the amount of infection present. If the bile is clear, I do not hesitate to close the common duct without drainage. All cases of pancreatic necrosis demand operation as soon as the

diagnosis can be established and the patient brought into condition for it. The necrotic tissue should be given a chance to extrude itself along a drainage tract, either through the peritoneal surfaces above or below the stomach or by drainage through the foramen of Winslow. If the

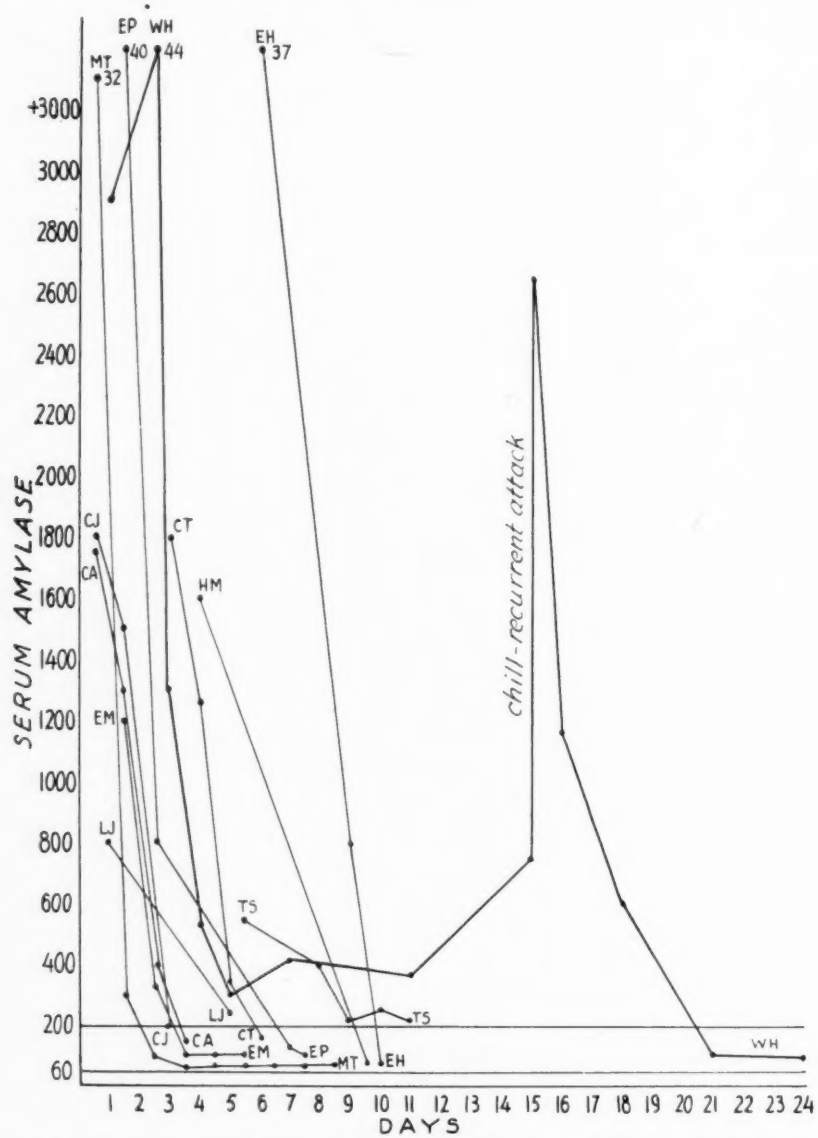

Fig. 2.—The serum amylase drops to the normal range within four to five days in most instances of acute pancreatic edema. In one patient a recurrence of an acute attack came before a drop to the normal range.

common duct is blocked it also should be drained. A certain number will be saved in this manner.

For the relief of intractable pain during the attack the opiates may be successful. Nitroglycerin at times relaxes the sphincter of Oddi

TABLE I

	DIED	PER CENT
Acute Pancreatic Edema		
70 Cases	11	15.71
41 Cases with operation	11	26.82
29 Cases without operation	0	0.00
Acute Pancreatic Necrosis		
27 Cases	19	70.37
18 Cases with operation	10	55.55
9 Cases without operation	9	100.00

and gives dramatic relief (Elman[10a]). Paravertebral injection in the eighth to tenth dorsal segments has been advocated (Popper[26a] and Dinsmore and Nosik[9]).

Large meals and alcoholic bouts should be avoided in the period after leaving the hospital. In some cases mild pancreatitis attacks come on even after biliary tract surgery in these patients. Elman[10a] gives his patients tablets of 1/100 gr. (0.0006 Gm.) glyceryltrinitrate to carry with them. If one of these tablets is put under the tongue promptly at the beginning of an attack it may be prevented.

Results.—Review of the current literature shows that any large series of acute pancreatitis cases carries a considerable mortality. Thus, Fallis[11] had 46.2 per cent, Brütt[4] 50.0 per cent, Morley[22] 35.3 per cent, Yates[22] 50.0 per cent, Ogilvie[22] 50.0 per cent, Griessmann[15] 34.0 per cent, Carter and Hotz[5] 53.3 per cent, Connell[8] 36.3 per cent, Lampson[19] 33.0 per cent, and Reichl[28] 46.23 per cent, an average of 43.43 per cent for these reports. In our previous collected results for immediate operation out of 911 cases, there were 447 deaths or a mortality rate of 49.06 per cent.[23a] The combined figures show only a slight improvement, 46.24 per cent. Our own figures show 67 cases in which operation was done, with 24 deaths, or 35.8 per cent.

As long as surgeons operate without using the amylase test to help in their diagnosis, there will be a mortality from acute edematous pancreatitis. This should not be, but it accounted for two deaths in our series of 19 cases after the amylase test was available. Previous to that our mortality was 9 out of 22 cases in this group when operation was necessary to decide what type of disease we were dealing with. When

TABLE II

	DIED	PER CENT
Acute Pancreatic Abscess		
11 Cases	6	54.54
8 Cases with operation	3	37.50
3 Cases without operation	3	100.00
Postoperative Pancreatitis		
20 Cases*	15	75.00

*Before 1939, post mortem was the only way to know, therefore others may have had it; now it can be tested.

the test was used properly we treated 29 cases of pancreatic edema without a death, not operating during the height of the reaction.

CASE REPORTS

R. D. (Hospital No. 217849), a 52-year-old well-developed and well-nourished man was admitted to the hospital, being referred by his doctor, with a diagnosis of perforated gastric ulcer. He was seen in the emergency department by numerous observers who all agreed on the diagnosis. His history was as follows: Fourteen hours previous to admission he was awakened by epigastric pain. Three hours before it became much worse, with radiation to his back. The pain was relieved by morphine. He had a vague gastrointestinal history previously; taking soda had brought relief. On entering, his temperature was 36.8° C., pulse 82, respiration 24, blood pressure 130/90, white blood cells 11,000. His abdomen was flat. There was boardlike spasm in the upper quadrants and spasm in the flank on the right. No peristalsis was seen. Flat plate of the abdomen showed no free air. I was asked to see him before I went home and agreed with the diagnosis. I asked that the operating room be set up in preparation for a perforated ulcer operation, but I suggested that an amylase test be done just as a matter of interest. By the time I reached home there was a telephone message saying that his amylase test was 6,400. Needless to say, operation was not performed. The next day his amylase was 2,100; it gradually subsided over the next week and he went home following this against advice, because he was a farmer who wanted to get his crops in and he would not stay for study, gall bladder series, etc.

S. R. (Hospital No. 178017) was a tremendously corpulent, 39-year-old woman who had had recurrent attacks of gall bladder disease. Twelve days before admission she had a severe attack associated with dull pain, nausea, and vomiting. Morphine and atropine failed to relieve her. She had right upper quadrant tenderness and spasm in the gall bladder area. Blood count was 32,000, icterus index 18, and blood amylase 1,600.

The attending surgeon thought that she was in urgent need of operation, the diagnosis being cholelithiasis, stone in common duct. Cholecystectomy with choledochotomy was done on the day of admission. The patient died two days after operation. At operation she had marked edema of the pancreas as well as gall bladder infection.

S. K. (Hospital No. 183524), a 65-year-old well-nourished man, was on the medical service, being studied because of right upper quadrant pain. Gastrointestinal series showed duodenal ulcer, and gall bladder test showed poorly functioning gall bladder. While still in the hospital he had a sudden onset of acute, persistent, severe, pain and spasm, vomiting, marked tenderness in the upper abdomen, most marked in the epigastrium. His abdomen was rigid, less so in the lower than in the upper. Liver dullness was present. There was rebound tenderness in the epigastrium. Temperature was 38° C., pulse 130; white blood count went from 12 to 16,000. Blood pressure was 190/85. No flat plate of the abdomen was taken for free air but a diagnosis was made by three surgeons, who saw this patient, of perforated duodenal ulcer. He was taken to the operating room and under vinethene and ether, free fluid and swelling of the entire pancreas with edema were noted. The abdomen was closed. The patient died the second day following operation.

Post-mortem examination showed acute pancreatitis with fat necrosis and peritonitis. From the fluid removed at operation amylase test showed a reading of 1,070.

E. M. (Hospital No. 134723), a very obese bartender, 34 years old, and a chronic alcoholic, was admitted to the hospital for the sixth time with a diagnosis of chronic cholecystitis, stones, and nephritis. He had continued attacks of nonradiating

epigastric pain, nausea, and vomiting. Three days before admission these atttacks were unusually severe in the right upper quadrant with epigastric pain and constant cramps, accompanied by vomiting. There was no relief from opiates. Temperature was 37.6° C., pulse 100, respiration 30, blood pressure 200/115. He was a tremendously obese man. There was acute tenderness in the epigastrium, but also in the lateral wall, especially over the gall bladder. His liver was three to four fingerbreadths below the costal margin. White blood count was 16,600, icterus index 13. He continued to have pain with no relief, and repeated vomiting. He called out in pain and drank a large amount of water. He went into shock, became unconscious. Medical men considered this to be a case of diffuse peritonitis with empyema of the gall bladder. Surgical consultation was called. It was suggested that pancreatitis might be a possible diagnosis. Accordingly, he was tapped. Blood-tinged fluid was obtained which gave an amylase reading of 400. That night he vomited and aspirated the vomitus and died.

Post-mortem examination showed acute pancreatitis, aspiration pneumonia, chronic cholecystitis, and cholelithiasis.

M. K. (Hospital No. 161450), a slightly obese, 22-year-old woman, had some pork chops for dinner. Twenty-four hours later she had severe epigastric pain radiating to the back, low down. It was agonizing and boring in character. She was nauseated but did not vomit. She was in acute distress, rocking back and forth in pain, her arms pressed to her back. There was a boardlike spasm of the whole abdomen and marked tenderness over the gall bladder. There was slight distention and some spasm to the left. Murphy's sign was positive. Temperature was 37.6° C., pulse 84, respiration 20, white blood count 17,000, amylase 2,000. She was given 75 r. units of x-ray treatment. During the night she vomited a small gallstone. The next day she felt much better. Her amylase test twenty-six hours later was 230. Five days after this, cholecystectomy with exploration of the common duct was done. The gall bladder showed chronic cholecystitis with one small stone. There were no stones in the common duct. She was discharged well.

L. B. (Hospital No. 2361), a very obese, 49-year-old woman, was admitted to this hospital previously in August, 1932, with a history of nausea and vomiting. In October, 1932, she had nausea and vomiting, dizziness, and dull pain in the upper abdomen. This was increased during vomiting. There was general abdominal pain and tenderness, more in the right flank, slightly more in her epigastrium. Temperature was 38.8° C., pulse 108, respiration 24, blood pressure 126/80. A diagnosis of acute pancreatitis was made. Exploration was carried out. Stones were found in the gall bladder. The pancreas was indurated and there was an abundance of fluid. Cholecystostomy was done.

The patient was near death following operation, but after twelve hours she made a rapid improvement and the tube drained for forty-five days, after which she was discharged. She was to come back for cholecystectomy later the same year. An appointment was made for Nov. 20, 1932. She did not appear at this time, however, and she felt well. She continued working as a school nurse. Nine days previous to this admission, June 12, 1940, she had an upper respiratory infection and three days before, crampy, abdominal pain, and much gas on the stomach, relieved by enema and vomiting. Temperature was 37.6° C., pulse 85, respiration 18, blood pressure 115/88. She was apparently in no distress. There was a subsiding pharyngitis. Abdomen was soft with no spasm. There was a slight resistance to deep palpation on the right of the upper abdomen. White blood count was 16,000. Flat plate of her abdomen showed nothing. Amylase test was 2,100. Oral cholecystogram showed no gall bladder shadow. She was given intravenous fluids and felt much improved; in the course of three days the condition subsided to normal. She was discharged without operation although it should be carried out, I believe. This is a good case because it is a two-times proved acute pancreatitis.

L. P. (Hospital No. 27793), a 25-year-old, obese Negro woman, was admitted to the hospital April 1, 1942. Two hours before she had severe crampy abdominal pain associated with nausea and vomiting. Two weeks before she had crampy abdominal pain associated with her periods, which was usual. Her two last periods had been of unusual length, eight to nineteen days instead of the usual three to four days.

Two hours before she had severe crampy abdominal pain, nausea, and vomiting without radiation of the pain, although some of it was in her left shoulder. Temperature was 37.4° C., pulse 72, respiration 18, blood pressure 110/60. She did not seem to be in any very acute distress. There was marked tenderness to deep palpation in the epigastrium but no distention or spasm. There was a mass in the right side close to the uterus and the white count was 32,000. The diagnoses suggested were many: cholecystitis, penetrating peptic ulcer, ruptured ectopic pregnancy, ruptured follicle, hemoperitoneum, and acute pancreatitis. Acute pancreatitis was diagposed by the fact that her amylase registered 400 and a cul-de-sac puncture was made which gave 2,500 c.c. of bloodstained fluid. Amylase on this was 1,800. Flat plate of her abdomen showed gallstone shadow, probably.

For one week she was treated by small doses of radiation therapy and her amylase dropped to the lower limits of normal. On April 14 it was decided that she had a left subphrenic abscess and this was drained, the pus pocket being identified. Fat necrosis was demonstrated in the tissue and necrotic fat from the drainage area. She was placed on Wangensteen suction. Intravenous fluids were given and she had a stormy course, draining profusely and developing glycosuria. She was given sulfonamide drugs and the condition gradually cleared up; on June 29 the wound was closed and there were no complaints. We have not been able to contact her since she left the hospital.

E. M. (Hospital No. 82365), a 53-year-old well-developed and well-nourished woman, was admitted to the hospital May 16, 1943. Twenty-four hours previously she had a sudden onset of very severe mid-abdominal pain over the gall bladder, radiating to the back, shoulders, and around to both sides. She vomited repeatedly. At entry she was very ill and pale and had general tenderness with spasm, increased in the right upper quadrant and epigastrium. There was rebound and referred tenderness in the right upper quadrant. Temperature was 37° C., pulse 108, respiration 24, and blood pressure 105/50. Icterus index was 8, blood amylase 3,200.

In 1912 her gall bladder had been drained and she had many attacks of No. 3 pneumococcus infections from that time on. A diagnosis of acute pancreatitis and chronic cholecystitis and cholelithiasis was made. Her amylase subsided over the next week so that it was within normal range. On May 31 a cholecystectomy with drainage and dilatation of the common duct was carried out. A fibrous constriction of the common duct was found. There were no stones in it, although the gall bladder contained three. The common duct was drained with a catheter which was taken out June 5, 1943.

The patient has remained well following this.

E. O. (Hospital No. 164872) was admitted to the emergency department May 6, 1940. While in church the day before, this 49-year-old woman had sudden pain in the right upper quadrant just below the costal margin. The pain became more severe that evening and radiated strongly to her back. She had fever and chills. The pain was constant, was not relieved by induced vomiting, but was somewhat relieved by hypodermic injection. On entry her temperature was 40° C., pulse 104, respiration 24, and white blood count 23,400. She was acutely ill, pale, and slightly jaundiced. Her extremities were cold and sweaty. There was deep tenderness in the right upper quadrant and none elsewhere. Her urine showed positive bile. Amylase was 50 on admission, icterus index 15. After two comfortable nights she

had severe epigastric pain and distention and boardlike spasms in both upper quadrants. There was marked tenderness in the right upper quadrant, some in the left upper quadrant, but no radiation. Her vital signs were up again. This time amylase reached 1,800 and the icterus index was 27. Her left pupil had been dilated since admission and was noted to be so still. The next day amylase was 2,300 and the icterus index 30. She was given radiation treatment consisting of two doses of 100 r. and 75 r. each and there was immediate subsidence of the amylase reaction down to 200 by noon of May 10. The icterus index dropped to 10. On admission her diagnosis had been a question of pneumonia or of acute cholecystitis, but the positive amylase test when she had the attack in the hospital made it apparent that we were dealing with an acute pancreatitis. After hydration and transfusion she made a marked clinical improvement and nine days after admission cholecystectomy was done with drainage of the common duct. The gall bladder showed acute and chronic cholecystitis without stones. The pancreas gave evidence of acute pancreatitis. It was enlarged to twice its normal size, firm, edematous, and had a bilious discoloration to it. Convalescence was uneventful and she was discharged on the fourteenth day. There was no follow-up study.

E. P. (Hospital No. 177924) was admitted to the emergency department May 14, 1941. She was a 48-year-old woman who had a sudden onset of severe epigastric and right upper quadrant pain. The pain radiated to the left and through to her back with some pain in the right shoulder. This was accompanied by nausea and vomiting of undigested food. Since her entry she has had continued severe, unrelieved pain, nausea, and vomiting. She had a similar attack three weeks before, but it was milder in nature. In her past history she has had much epigastric distress, burning, and gas following meals. There was also some fat intolerance.

On examination, temperature was 37° C., pulse 84, respiration 22, blood pressure 110/70. She was a very obese woman in acute abdominal distress. There was generalized spasm of the abdomen and extreme epigastric tenderness in the right upper quadrant with spasm. Referred and rebound pain to the right upper quadrant and periumbilical region. There was a positive Murphy's sign, no costovertebral angle pain or tenderness. White blood count was 13,800, icterus index 16 units, and amylase 4,000. Flat plate of the abdomen was negative.

She was confined to bed and treated with fluids parenterally with subsequent fall in the blood amylase level to 94. Her icterus index fell to 9 units. She was discharged May 26 to stay on a surgical soft and low-fat diet, with a diagnosis of acute pancreatitis and questionable acute cholecystitis. June 16, 1941, she was given an intravenous cholecystogram which showed a nonfunctioning gall bladder. A diagnosis of chronic cholecystitis was made. It was decided to operate upon her in the fall at her request. She remained symptom-free on a diet until three days before her second admission, which was Dec. 26, 1941. The appointment for operation in the fall was not kept, but on the date of this admission she had an acute mid-upper abdominal pain with radiation to the back, associated with nausea but no vomiting. The attack lasted for about two hours. She had a similar attack on the day prior to admission and on the evening of admission had the third attack. She thought these attacks were brought on by taking some cream with her food. Cholecystectomy and exploration of the common duct were carried out. An acute and chronic gall bladder, and cholecystitis with cholelithiasis were demonstrated. The gall bladder was packed full of hundreds of small faceted stones. Following operation she has done very well and has remained symptom-free to date.

M. R. (Hospital No. 205096), a 52-year-old obese, Irish woman, was admitted May 20, 1943. She gave a five-year history of head pain and indigestion with two acute attacks accompanied by nausea, vomiting, and acholic stools. Following a fatty meal she had onset of intermittent epigastric pain localized in the right upper

quadrant with no radiation. There were no chills or fever. Nausea and vomiting were present. Bowel movements were white, urine was black, and she was blown up with gas. Temperature was 37.8° C., pulse 100, and respiration 20. She was moderately acutely ill. There was tenderness in the right upper quadrant and epigastrium, positive Murphy's sign, no costovertebral angle tenderness, and but slight icterus. White blood count was 8,300, blood pressure 148/90. She had bile in the urine. Icterus index was 18 and an amylase which was done three days after admission was 600. She was treated symptomatically with a low-fat diet and bed rest. Her pain and tenderness subsided and she was to return later for a cholecystectomy as the gall bladder test showed stones in the gall bladder and calcification in the gall bladder wall.

She entered the hospital again Oct. 5, 1943, with severe pain which began at midnight in the epigastrium, radiating to the back and lower ribs, continuous in nature, associated with nausea and vomiting. This was the fourth of similar attacks. Icterus index at this time was 15 and her amylase 800. This apparently was a recurrence of an acute pancreatitis. After the amylase had subsided to a normal level she was operated upon. Cholecystectomy was done on October 12. The gall bladder was full of stones and there were some in the common duct. The pancreas was chronically thickened and hard. A diagnosis of chronic pancreatitis was made. The common duct was explored and drained. The tube was removed on Oct. 22 and the wound was healed by November 2.

C. M. (Hospital No. 156824) was admitted Dec. 28, 1939. A 29-year-old, moderately obese woman had, five days before admission, a sudden severe sharp pain in the upper abdomen, on both sides of the epigastrium, but more on the right. This pain passed off after a few minutes, leaving distention and fullness. The attacks were intermittent until the day of admission, when they became extremely severe and constant. There were no radiation, no nausea, vomiting, nor jaundice. Her bowels were regular. Temperature was 37.2° C., pulse 120, respiration 20, blood pressure 100/60. She looked distressed, flushed, and acutely ill. She had a short, choppy breathing and when she took a deep breath she caught herself. She cried out with pain present on both sides. There was general abdominal tenderness, very marked over the entire upper abdomen and both flanks. There were generalized spasm, rigidity, and distention. White blood count was 14,500, amylase 2,000. Her temperature went to 39° C., and pulse to 150. She was given four treatments with x-ray, two of 100 r. and two of 75 r. each, so that by January 1 her amylase was recorded as 100 and the day after the first treatment the acute pain and distress subsided. A nasal tube was inserted for several days. For one week her condition remained precarious. Then she made a dramatic improvement and her temperature came down to normal. The pulse lowered to around 100. Symptomatically the patient showed a marvelous improvement. She was given parenteral fluids and felt quite well for another week.

At about this time, however, her temperature became elevated to approximately 38° C. each day. Her pulse fluctuated markedly. Jan. 13, 1940, she suddenly went into mild shock with cold, clammy extremities. Pulse went to 160. Respiration was rapid and shallow. Blood pressure went down to 80. At times it was not obtainable. She was given treatment for shock and made a slight improvement. Examination showed a large mass in the left upper quadrant and epigastrium, about 20 by 10 cm., hard, fixed, and slightly tender. Under local anesthesia a pancreatic abscess was drained. Amylase test showed 1,500 units but it gradually subsided down to 130. She showed occasional sugar in the urine but made very little progress and on the fifteenth postoperative day she passed a bloody stool. On the seventeenth postoperative day she had a hemorrhage from the wound. Her course from this time on was rapidly downhill and she died Jan. 31, 1940, slightly over a month after admission.

Post-mortem examination showed a peripancreatic abscess, a retroperitoneal abscess, a perforation of the colon next to the abscess, and a small stone in the common bile duct, with the wall of the abscess containing many bowel loops, chronic pancreatitis, and fat necrosis over the peritoneum.

J. S. (Hospital No. 176444), a 69-year-old obese woman, was admitted to the hospital April 9, 1941. She had severe pain in her back and abdomen over a period of the last five years, coming on approximately once a month and lasting four to five days, then disappearing spontaneously. Five days previous to admission she had severe pain in the lower abdomen which she had never had previously. This persisted until her admission, keeping her awake at night. She said the pain was greater in the right lower quadrant but was indefinite as to its situation. Associated with this there was also marked back pain which had been constant. The patient had been confined to bed. There had been no nausea, vomiting, constipation, injury, genitourinary symptoms, or gall bladder history. Her appetite had been poor and she had taken mostly liquids, with the result that she has lost about twenty pounds in the last year.

On entry her temperature was 37° C., pulse 100, respiration 22, and blood pressure 115/70. She appeared to be in considerable pain. Her skin was dry and flushed. She showed generalized arteriosclerosis and arteriosclerotic heart disease, class 2-A. Her abdomen was rounded, diffusely tender with spasm and marked tenderness at the dorsal-lumbar spine, both sides. No masses or viscera were palpable. There was a question of fluid in the abdominal cavity and marked distention. A tender mass was thought to be felt in the right upper quadrant. She showed mild diabetes. While blood count was 17,000. Amylase test was done and showed 130. Icterus index was 8, nonprotein nitrogen, 133, and a flat plate of the abdomen was negative. There was little from which to make a diagnosis, according to the consultant who attended, but it was suspected that the patient had an acute pancreatitis in spite of the low amylase test.

Two x-ray treatments of 100 r. each were given over the pancreas. Under local anesthesia a large amount of prune juice fluid was obtained. A normal gall bladder was noted. There were fat necroses in the omentum, one piece of which was biopsied and showed fat necrosis microscopically.

She was treated with parenteral fluids and insulin with a good response. Her postoperative course was marked by daily elevation of temperature and one episode of decompensation of her heart which required digitalis for control. She continued to be lethargic for a couple of days postoperatively. Her course was fairly good although a large tender mass was present in her abdomen which was considered to represent pancreatic abscess. On the twenty-sixth day she had acute abdominal pain and the passage of clotted blood per rectum and coffee-ground vomiting. The patient went rapidly into shock and did not respond to therapy. She died on the twenty-sixth postoperative day, or exactly one month after admission.

At post-mortem she showed acute hemorrhagic pancreatitis with extensive fat necrosis, perforation of the duodenum, and transverse colon. There was a perinephric abscess on the right side and chronic cholecystitis and cholelithiasis with impacted stones in her cystic duct.

A. F. (Hospital No. 180482) was a 65-year-old, well-nourished man. While walking, he had a sudden, severe pain in the pit of his stomach. It spread through his entire abdomen. He got relief by lying on his left side and remaining quiet. This pain was of twelve hours' duration when he was admitted to the emergency department. He was vomiting a coffee-ground material continuously if he took anything by mouth. He was pale, in acute distress, perspiring, and anxious. There were slow, thready pulse, diffuse tenderness of the abdomen, spasm, and rebound tenderness. There was no distension although liver dullness could not be definitely made out. A flat plate of the abdomen showed no free air and no trapped air in terminal

loops. Temperature was 38° C., pulse 80, respiration 24, blood pressure 120/80, icterus index 20, and amylase 2,600. His referring diagnosis was ruptured peptic ulcer.

He was given four treatments of 100 r. each by x-ray and his amylase dropped from 2,600 to 1,280. He made a steady improvement. His pain disappeared after the third day. He was well hydrated, comfortable, and on a house diet. On the fifteenth hospital day he had an attack. His temperature went to 40.5° C. Amylase at this time was 1,778, icteric index 36. He had crampy pain. This was undoubtedly an attack of recurrent pancreatitis. He was treated again as previously. He became afebrile, his icterus subsided, and one week later an intravenous cholecystogram was done, following which he had a steady downhill course and died on the thirtieth hospital day. Post-mortem examination was not obtained.

C. V. (Hospital No. 35226), a 48-year-old well-nourished woman who was a pernicious anemia patient, was admitted to the hospital. Fifteen hours before, she had upper abdominal pain which was the fourth and severest attack of this kind in the last year. There were persistent vomiting of bilious material and pain which ran from the left to the right. Thirty years before she had jaundice. Her vital signs were normal. She had epigastric tenderness and moderate spasm, more prominent on the right than on the left side. There was a positive Murphy's sign. Blood pressure was 160/88, white blood count 13,000, amylase 1,200, icterus index 16. Without waiting for the result of the amylase test, operation was carried out. Serosanguineous fluid was present in large amounts. Gall bladder was thin. There were no stones. Pancreas was enlarged, edematous, and had a yellow-greenish discoloration. Amylase on the abdominal fluid was 2,700. Following operation the patient was given x-ray over the pancreas and the amylase test dropped to normal within three days; she was discharged on the fourteenth day in good condition.

In August, 1941, she had an attack which was similar to this and went to a hospital in Auburn where a former resident of this hospital worked. He made a diagnosis of an acute pancreatitis. The attack subsided in four days and she was discharged from the hospital. She has been followed in the pernicious anemia clinic since that time and was well to October, 1944.

J. S. (Hospital No. 120402), a 62-year-old rather obese woman, Oct. 12, 1936, had a definite attack of acute pancreatitis diagnosed by operation. Fat necrosis was present at that time, as well as edema of the pancreas. Since then she has had gas after eating regularly. Three hours before admission, she had an acute abdominal pain localized in the upper abdomen, severe, generalized associated with nausea but no vomiting. She was in acute distress, appeared to be in shock. She had cold, clammy extremities. There was slight distention. Tenderness was general and there was spasm in the right upper quadrant. The liver dullness was not obliterated. Temperature was 36.6° C., pulse 84, respiration 24, blood pressure 115/68, icterus index 18, white blood count 25,000, and amylase 4,000.

She was given 300 r. x-ray treatment over the pancreas and the following day her amylase had dropped to 1,200 and by the third day was in the range of normal. Her tenderness subsided but distention remained. She was allowed up and gradually became normal. A gall bladder series was taken December 10 and showed stones in the gall bladder. Cholecystectomy with exploration of the common duct was done. There were visible fat necroses present and the pancreas was seen to be firm, rough, and nodular. She responded well to operation and was discharged home well. She has been well to date. This woman has had two attacks of acute pancreatitis.

F. B. (Hospital No. 173081), a 40-year-old obese woman, was admitted to the hospital complaining of sharp pain in the shoulders and back and between the shoulder blades, of five weeks' duration. She had pains in the chest for the last five weeks, also with persistent cough. She had para-umbilical pain for five weeks

and two days before admission it became very sharp, something having "let go" in her left abdomen. She had severe sharp pain after that, vomiting, hot burning pain, and diarrhea. In her past history she had a bad heart, rheumatic heart disease. Temperature was 38.6° C., pulse 96, respiration 20, white count 10,000. She was in acute pain. There was generalized abdominal tenderness which was increased to the left of the umbilicus and in the left upper quadrant there was a firm, herniated mass at the umbilicus, tender to touch. She kept her left leg flexed for relief. A diagnosis of incarcerated omentum and strangulated hernia was made.

Under vinethene and local anesthesia the hernial sac was opened. A strangulated omentum, greatly thickened and hemorrhagic, was dissected away from the colon. For six days she was afebrile and comfortable. Then she developed fever and moderate pain again. There was moderate distention, tenderness and pain in the right upper quadrant, moderate umbilical pain and muscle spasm, and free fluid which was sterile to culture. The question of pancreatitis was raised. For one month she had fever and pain which was severe and constant, finally slowly improving. About three-fourths of the way along her course an amylase test was taken which showed a reading of 540 and repeated tests showed it never below 420. Microscopic examination showed acute and chronic organizing inflammation of the omentum and sterile fluid. She was discharged one month after entry in good condition. This was undoubtedly the result of an acute pancreatitis with resolution of widespread inflammatory reaction.

M. T. (Hospital No. 125461), a 48-year-old slightly obese woman, was admitted to the emergency department April 10, 1941. She had sudden, severe, epigastric pain starting four hours previously, radiating to her back and to the left. She had two spells of vomiting. The pain went to the left costovertebral angle also. Her symptoms suggested "intestinal obstruction." Temperature was 37° C., pulse 86, respiration 24, blood pressure 150/95. She was uncooperative and complaining, rolling about in bed and moaning. Her abdomen was large and rounded. There was no free fluid present. She had tenderness at the left costovertebral angle and diffuse abdominal tenderness. There was no spasm or mass at entry. Her icterus index was 7, white blood count 16,000. The diagnoses suggested were: gall bladder infection, posterior perforated ulcer, partial intestinal obstruction, kidney stones, pancreatitis, ovarian cyst. She was admitted to the hospital and her amylase test showed 3,200. A course of x-ray therapy was given to her, consisting of 100 r. units, six times. Coincident with this treatment there was a rapid drop in her amylase from the initial figure to 80. She refused operation. After the condition had subsided, she was in the hospital for about one month, and during that time a mass was palpable in her right upper quadrant. This was thought to be either a distended gall bladder or perhaps an abscess in the region of the pancreas. However, she continued to refuse operation and was discharged in the latter part of May, 1941. At the last report the patient still refused to accept advice but was having considerable indigestion and symptoms of chronic cholecystitis in attacks. She was last seen in April, 1942, at which time she remained about the same as she had been previously.

CONCLUSIONS

Pancreatitis must be considered as a possibility in patients complaining of sudden severe epigastric pain.

The serum amylase test is of the greatest assistance in deciding whether the pancreas is involved.

The test is easily carried out, is accurate, and gives more important information than any of the routine laboratory tests.

Clinically, there are two distinct pathologic types of acute pancreatitis, acute edematous and pancreatic necrosis.

Acute edematous pancreatitis can be diagnosed by the serum amylase test and the rapid improvement under conservative treatment.

Pancreatic necrosis must be suspected when the patient fails to make improvement within a few days.

In acute edematous pancreatitis operation should be deferred until the reaction has subsided.

Acute edematous pancreatitis is followed by chronic pancreatitis frequently, as is demonstrated at late operation.

Pancreatic necrosis is followed by abscess, diabetes, and pseudocysts in a proportion of those who survive.

Conservative treatment of pancreatic necrosis or pancreatic abscess is disastrous.

When diagnosis of either condition is suspected, operation is indicated as soon as the patient can be properly prepared for it.

Biliary tract disease should be treated after an acute pancreatitis attack if it has played a part in the onset.

Any surgical manipulation about the lower end of the common duct or the head of the pancreas is likely to be followed by postoperative acute pancreatic edema.

This can be demonstrated by the amylase test.

There is a considerable danger of this in the resection of posterior duodenal ulcer perforating into the pancreas.

It carries a mortality which should be taken into consideration in any series of duodenal ulcer resections.

The case reports were selected to illustrate points made in this discussion.

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INVESTIGATION OF THE CAUSE AND PREVENTION OF GAS PAINS FOLLOWING ABDOMINAL OPERATION

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IN THE immediate convalescence from the routine type of abdominal operation the most common causes of distress are gas pains, nausea, and vomiting; they are of such usual occurrence that they are accepted as a matter of course and little is done to alleviate them. Both distressing conditions can be prevented to a great degree by proper procedure. For a time after abdominal operations there is decreased motility of the bowel; therefore, any gas in the bowel remains there, causing distention and pain.

In a group of patients at the University Hospital a few years ago, observations on the motility of the bowel after appendectomy were conducted by one of us (H. H. D.). In a series of cases in which appendectomies were performed under spinal anesthesia, each patient was given a small quantity of barium a few days before operation and its passage through the intestinal tract watched. The same quantity of barium was given immediately after operation and its progress observed. At the end of two hours the stomach was empty in 64 per cent of the patients in the control group, and it was empty in only 20 per cent of the patients in the operative group. At the end of twenty-six hours, we found that usually the barium had entered the rectum in the patients in the control group; it was in the ascending and transverse colon in those of the operative group. At the end of two days the stomach was empty in the patients in the control group, but the barium was in the transverse colon and the sigmoid in those of the operative group. It was evident that a definite decrease in peristalsis followed even a simple appendectomy.

Puestow,[1] in his work on peristalsis, showed a contrary motility between the small and large bowel, that is, the small and large bowels were not stimulated by the same drugs. He found the small bowel was stimulated, but the colon was inhibited by opiates, physostigmine, prostigmine, methylsulfate, and choline derivatives. Pitressin stimulated the colon but inhibited the small bowel.

In the past there has been an erroneous belief that the intestinal gases were the result of putrefaction arising in the bowel. In 1918, as a result of his experiments with animals, Kantor[2] showed that swallowed air is the main source of gas in the bowel.

In 1926, McIver, Benedict, and Cline[3] analyzed gases withdrawn from the rectum in postoperative patients with distention and in nonsurgical

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patients. They found that the percentage of nitrogen was uniformly high. Since free nitrogen is liberated in the intestines in small quantities, if at all, it must be derived from air, 79 per cent of which is nitrogen.

In 1934 Hibbard and Wangensteen[4] analyzed the composition of gas in the patient with distention and found that it was: carbon dioxide 6 to 12 per cent, oxygen 10 to 12 per cent, methane and hydrogen low, and nitrogen 70 per cent. Hibbard and Wangensteen concluded that 68 per cent of the gas in the bowel was due to swallowed air. In 1942, Singleton, Rogers, and Houston[5] conducted a similar experiment and found: carbon dioxide 9.51 per cent, oxygen 5 per cent, methane 1.2 per cent, and nitrogen 84 per cent. The source of the nitrogen was considered to be swallowed air.

Wangensteen and Rea,[6] in 1939, noted that with cervical esophagostomy there was no excess gas in the obstructed intestines. It has been noted, also, that in patients with complete esophageal strictures abdominal operations resulted in no gas in the bowel, and there was no tendency toward distention.

Much of the air which is swallowed is done so in the induction phase of anesthesia. Were the patient not under the influence of sedatives prior to induction, the amount of air swallowed would be much higher. Uneasiness, excitement or resistance on the part of the patient is conducive to the swallowing of air.

Proceeding upon the hypothesis that the swallowed air is the cause of gas in the bowel, we carried out a series of clinical experiments at the Immanuel Hospital.

The Levin tube was inserted one-half hour after the patient had received a preoperative hypodermic of pantopon, gr. ⅓, and scopolamine, gr. 1/100. Thus the patient was semiconscious and relaxed and did not resist. Patients have been taken to the operating room and returned, not knowing that the tube had been inserted and was functioning. In the operating room the Levin tube was connected with the bottles for gastric suction. By measuring the amount of water displaced in the bottle by the Wangensteen technique, the amount of air swallowed by the patient was determined.

One of the problems encountered concerned the length of time the tube should be retained by the patient. The average of sixty-four hours (See Table I), we found to be too long. This average resulted because of experimental attempts to find the ideal time for removal, the span being from twenty-four to seventy hours, or longer. Careful attention to the Wangensteen fluid balance helped to determine the ideal time for removal. At first, more fluid came back by Wangensteen suction than the patient swallowed, but during the first, second, or third day, depending on the patient, we found that the patient was drinking more than the suction could remove. This indicated that the material was going past the pyloric sphincter. The character of the stomach contents changed

TABLE I

	NUMBER OF PATIENTS	C.C. AIR DURING INDUCTION	AV. AIR DURING INDUCTION	C.C. AIR DURING OPERATION	AV. AIR DURING OPERATION	TOTAL AIR SWALLOWED	AV. AIR SWALLOWED	NUMBER HOURS LEVIN TUBE	AV. NUMBER HOURS LEVIN TUBE
Pelvic operations	14	3200	228.57	2500	178.57	5700	407.14	774	55.29
Cholecystectomies	14	800	57.14	1900	135.71	2700	192.86	824	58.88
Resection and Colostomy	5	250	50.00	1150	230.00	1400	280.00	444	74.00
Miscellaneous	12	1650	137.50	1775	148.08	3425	285.42	815	68.00
Appendectomies	8	850	106.25	265	44.25	1115	150.50	874	62.44
Totals	53		126.42	143	143.23		269.65		64.09

The miscellaneous here are all intraperitoneal surgery.

at this time. During the first hours following surgery there was a considerable amount of bile returned, but at the time more fluids were taken than returned by Wangensteen suction, the stomach contents returned clear, no longer bile stained, which indicated that there was no regurgitation from the duodenum. The time to remove the tube was indicated when a positive fluid balance, with no bile in the stomach contents, was achieved.

In the series included in this experiment, one patient who had an appendectomy swallowed 700 c.c. of air during induction; there were no postoperative complaints. A cholecystectomy patient who swallowed 600 c.c. of air complained of some gas pains which were relieved with atropine. In a subsequent operation, a stenosis of the sphincter of Oddi was found which may have been a contributing factor to the postoperative distress. During anesthesia for hysterectomy, a woman swallowed 1,000 c.c. of air; she complained of gas pains on the second day following removal of the tube, which had been removed at forty-eight hours. She was given four doses of pitressin for relief of gas pains. Another patient in whom we loosened some adhesions for partial obstruction, swallowed 2,150 c.c. of air; there were no complaints following surgery. A woman with bilateral oophorectomy swallowed 1,900 c.c. of air, and she had no complaints. Another with a cholecystectomy swallowed 600 c.c. of air; she vomited nine ounces on the first postoperative day, but was drinking water faster than the Wangensteen suction could remove it. She drank a total of 8,000 c.c. of water on the first postoperative day. These patients were fairly comfortable and had no distention because the air was removed immediately rather than allowed to go down into the bowel. Once the air has passed into the bowel it is too late to get complete relief by Wangensteen suction.

Apparently the only disadvantage in the use of the Wangensteen suction is the slight discomfort experienced by the patient because of throat irritation due to the tube. However, in one patient, the tube was removed because of this irritation and within twelve hours the

patient asked to have it reinserted because of abdominal distress. From the patient's point of view there was less discomfort in the irritation of the tube than in the alternative stomach distention. It was evident that a rectal tube, which was used very frequently prior to the use of Wangensteen suction, would cause as much, or more, discomfort than the Levin tube, and was not nearly as effective.

Comparisons were made of results obtained in patients operated upon before and those operated upon after we started the routine use of Wangensteen suction. There were 100 patients in the control group. In Table II, the first figure indicates the results when no Wangensteen suction was used, and the second figure indicates results with the use of the Wangensteen suction.

TABLE II

OPERATION	NUMBER OF PATIENTS	OPERATING TIME (MIN.)	TOTAL HYPODERMICS	AV. HYPODERMICS PER PATIENT	DAYS WITH GAS	NUMBER PATIENTS WITH GAS	PER CENT PATIENTS WITH GAS	DAYS GAS PER PATIENT	DAYS DISTENTION	NUMBER PATIENTS WITH DISTENTION	PER CENT PATIENTS WITH DISTENTION	DAYS DISTENTION PER PATIENT
Pelvic operations	45	63.78	503	11.18	65	31	68.88	2.09	20	12	26.65	1.66
With Wangenteen suction	14	62.86	226	16.21	19	9	71.45	2.01	1	1	7.14	1.00
Cholecystectomies	27	50.63	408	15.11	61	21	77.82	2.81	15	5	18.45	3.00
With Wangensteen suction	14	57.85	184	13.14	13	8	57.02	1.61	3	1	7.16	3.00
Resection and colostomy	5	73.00	95	19.00	7	3	60.00	2.33	5	2	40.00	2.50
With Wangensteen suction	6	86.66	82	13.63	3	2	66.66	1.50	1	1	16.65	1.00
Miscellaneous	23	77.24	272	11.80	41	16	69.52	2.56	20	10	43.56	2.00
With Wangensteen suction	14	49.14	166	11.83	8	8	21.43	2.66	4	2	14.26	2.00
Total without	100	63.92	1278	12.78	174	81	81.00	2.14	60	29	29.00	2.07
Total with	48	60.24	658	11.63	43	22	45.53	1.95	9	5	10.44	1.80
Appendectomies	10	22.00	48	4.80	2	1	1.00	2.00	0	0	0.00	0.00
With Wangensteen suction	10	23.00	58	5.80	3	2	2.00	1.50	0	0	0.00	0.00

The operating time was essentially the same, about one hour. The number of hypodermics per patient averaged eleven to thirteen.

With the introduction of the Wangensteen technique there was a decrease in the number of days in which gas was present, having dropped from 174 days in 100 patients to 43 days in 48 patients. The percentage of patients with gas that had been 81 per cent, dropped to 45 per cent. The average number of days with gas pains had been 2.4 but dropped to 1.95, and the pains were much less severe.

With the Wangensteen suction 5 patients, or 10 per cent, were distended with gas. Of these, 4 had been operated upon for intestinal obstruction, either complete or partial, and had moderate distention prior to surgery. In the group without Wangensteen suction there was distention in 29 per cent.

It has been mentioned previously that as a bowel stimulant for the reduction of gas, prostigmine is specific for the small bowel and pitressin for the large bowel. Prior to the use of the Wangensteen suction these drugs were used extensively for the attempted reduction of gas and distention. Sixty-four doses of pitressin, one-half ampule per dose, were used in fifteen patients in a control group. Twenty-four ampules of prostigmine were used in 4 patients. Of these, 11 had no distention prior to surgery. The categories of those who had received the drug were: cholecystectomy, 6 patients; pelvic operation, 5 patients; exploratory celiotomy, 4 patients. After the use of the Wangensteen suction was adopted, only 2 patients were given pitressin. Of the twenty-seven doses administered, 1 patient with distention before surgery received twenty-five.

Analyzing our control series, we found that the Wangensteen suction was used in 25 patients. Eight of these patients had either bowel resection or gastrectomy. In the other 17 it was used in an attempt to reduce distention after it had once begun. We now believe this to be ineffective. It has been the practice of many surgeons to pass the Wangensteen tube if the patient had marked distention and had severe gas pains, but in those in whom the gas had passed from the stomach into the bowel only partial relief was achieved because the Wangensteen tube did not reach into the bowel. With the Wangensteen suction functioning prior to anesthesia, gas is drawn out of the stomach before it has passed into the bowel and distention is prevented. This is particularly true in cases of obstruction of the bowel. Therefore, the Wangensteen suction should be used for the prevention of gas and the Miller-Abbott tube for the reduction of distention once it has developed.

A marked reduction in the need for rectal tubes, flushes, and enemas was observed. Coller, in a discussion on the subject, made the following remark: "As far as I know, enemas do nothing but exhaust the patient and stimulate reverse peristalsis and increase distention, and the rectal tube does nothing but cause piles."[5]

It is impossible to tabulate the clinical response of the patient. However, it was noted that, comparatively, these patients made better progress, felt better, and required less attention.

Wangensteen suction usually is unnecessary in appendectomies and herniorrhaphies since there is generally a minimum amount of gas and distention. The routine use of Wangensteen suction in appendectomies has been discontinued, but if there are signs of peritonitis it is passed immediately.

CONCLUSIONS

Immediately following abdominal operations there is some paresis of the bowel wall so that gas is not passed along the bowel properly, to be expelled. Therefore, it collects, causing distention, pain, and, at times, vomiting.

Swallowed air is the main source of gas in the bowel following operations. It is advised to institute Wangensteen suction during the anesthesia and surgical procedure, as well as in the immediate postoperative period, to evacuate the gas as it enters the stomach, and so prevent its entrance into the bowel.

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GASTRIC RESECTION FOR CERTAIN ACUTE PERFORATED LESIONS OF STOMACH AND DUODENUM WITH DIFFUSE SOILING OF THE PERITONEAL CAVITY

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FOR many years there appeared to be a geosurgical problem in the surgical management of peptic ulcers. The ulcers in peoples of continental Europe seemed to differ in certain respects, particularly in response to therapy, from those of the peoples of English-speaking nations. American and British surgeons were convinced that they obtained satisfactory results from the conservative operations of simple excision, gastroenterostomy, and pyloroplasty; whereas the surgeons of continental Europe learned very early that these procedures gave unsatisfactory results in their patients, and for the most part, they abandoned them in favor of the more radical operations of resection. To account for this apparent difference, several explanations were offered, among them differences of temperament, custom, diet, and associated gastritis.

However, in recent years as the surgeons of this country have accumulated an experience with gastric resection for peptic ulcers, and have had an opportunity to compare the late results of this operation with those obtained by the conservative procedures, they have become convinced, as were the surgeons of continental Europe, that resection produces a much higher percentage of permanent cures with no significant increase in operative mortality.

For more than a decade many German and Russian surgeons have followed the same radical course in the treatment of acute perforated ulcers. They have done emergency resections in the presence of diffuse soiling in all but the poorer risk cases and with a mortality which seems almost incredibly low. As a matter of fact, the reported mortality from resection has been definitely lower than that from simple closure of the perforations. DeBakey, in 1940, in an exhaustive review of the literature, collected 2,392 cases in which resection was done with a mortality rate of 13.4 per cent, and 5,589 cases of simple closure of perforation with a mortality rate of 25.9 per cent. It must be borne in mind that those in which resection was done represented a selected group in which the poorer risks were excluded. Judine, in 1937, however, reported a series of 418 cases of which 331 were good-risk patients, treated by resections with a mortality of 7.8 per cent, and 87 were poor risks, treated by simple closure or simple closure plus gastroenterostomy with a mortality of 32.2, giving an over-all mortality of only 12.8 per cent. Two years later he reported his own remarkable experience with 937 cases of resection, with a mortality of 8.9 per cent. Even lower mortality rates have been reported. Mulleder and Neuberger reported 21 cases of resection with 4.8 per cent mortality, and Odelberg, 20 cases of

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resection with 5 per cent mortality. Mortality rates under 14 per cent have been reported by von Haberer, Paul, Hromada and Newman, Gerhardt, Koehl, Marine, Peters, Rieder, Sostegni, Steiger, and several others. There are a few unusually low mortality figures reported for small series of cases in which the patients were treated by simple closure; Tilton, 2 per cent; Graham, 2 per cent; Pich, 2 per cent; Nicholas, 3.6 per cent; Gilmour, and Saint, 4.7 per cent; and Black, 5.4 per cent.

The advocates of primary emergency resection contend that not only is the immediate mortality less and certainly no greater than that of primary closure, but that the operation is curative rather than palliative. There is a high incidence of recurrent ulcer symptoms following simple closure, contrary to general opinion. In DeBakey's large collected series of followed cases, gastrectomy resulted in long-term relief of symptoms in 90.6 per cent of patients so treated; simple closure gave relief in only 65 per cent, and in 16.9 per cent of the simple closures subsequent operations were necessary. Harrison and Cooper have reported recurrence of symptoms in 82.5 per cent of cases following simple closure and Parker found that subsequent operations were necessary in 34 per cent of the simple closures he analyzed. The follow-up studies of cases in which resection was done, reported by Paul, Marine, and Odelberg, have indicated permanent cure in 100, 100, and 87 per cent, respectively.

These comparative figures are significant not as a basis for choosing between the two procedures, but rather as evidence that resection may be accomplished in the presence of a chemical peritonitis with little or no increase of hazard in the hands of skilled resectionists. It is not a matter of making a choice between the two procedures and establishing this choice as a routine. I believe that both procedures have definite roles in the management of these emergencies. I believe that the immediate interest of most cases is best served by simple closure of the perforation and that the high incidence of recurrence is not a deterrent. Recurrent ulcers requiring a subsequent resection can be handled by elective operations with less risk.

There are a few cases, however, in which primary resection is either the operation of choice or is definitely imperative. It is the purpose of this paper to present these indications with illustrative cases. These indications are:

1. Perforated carcinoma of the stomach
   (a) Delimited-resectable lesions
2. Perforated peptic ulcers associated with recent or simultaneous gross hemorrhage
3. Perforated peptic ulcers associated with fixed pyloric obstruction
4. Recurring perforations
5. Perforations with insignificant soiling
6. Very early perforation in the very young

Probably under no circumstances should resection be undertaken unless the patient is in fair condition, preferably young, and the perforation of not over twelve hours' duration.

PERFORATED CARCINOMAS

Acute perforation of a carcinoma of the stomach into the free peritoneal cavity occurs very infrequently. I have had the unusual experience of encountering two cases at operation. The first one was reported in 1944.[2]

One hundred twenty years ago Laennec recorded in his case book an instance of perforation of carcinoma of the stomach with peritonitis. Since then the case reports which have appeared in the literature have been collected periodically. In 1935, Aird reviewed previous analyses and assembled seventy-one reported cases. To these he added one of his own and seven others from the Royal Infirmary at Edinburgh.

From a review of the literature since 1934, in collaboration with Overmiller, I have collected an additional 138 cases in which the diagnosis had been established at operation or post-mortem examination. In approximately one-half of these cases no operation had been performed.

The combined series of 217 cases gives an erroneous conception of the incidence of perforation. Unquestionably only a few of the total number of cases have been reported. A more accurate impression can be gained from several available surveys of case records and autopsy reports. In these the incidence varies from 2.8 to 6 per cent, as follows: Brinton, 21 perforations in a total series of 507 cases of carcinoma of the stomach; Ditrich, 7 in 110; Lang, 12 in 210; Toinon, 9 in 255; Jaisson, 6 in 135; McNealy and Hedin, 116 in 3,289, and Casberg, 7 in 247.

In only 7 of the 217 cases was it alleged that a correct diagnosis had been made prior to operation or post-mortem examination. More astonishing was the failure, in many instances, to recognize at operation that perforation had occurred in a carcinomatous crater instead of a benign peptic ulcer. The failure to recognize the presence of the carcinoma suggests that many of the carcinomas which perforate are small and relatively confined lesions presumably suitable for resection. This premise has been borne out at the autopsy table. In these cases there is, therefore, the possibility of considerable salvage of life.

From the study of 46 case histories collected from the literature, Chavannaz and Radoiévitch distinguished four clinical types: (1) cases with a sudden onset of severe epigastric pain and manifesting the typical signs of a perforated peptic ulcer, this preceded by a long or short history of dyspepsia; (2) cases differing from (1) only in respect to absence of previous dyspepsia; (3) cases with an insidious onset exhibiting signs of a gradually progressive peritonitis; (4) cases in which the

patients die of peritonitis and at no time have symptoms to direct attention to the abdomen. About 70 per cent of the cases are types (1) and (2).

Records of gastric analyses were presented in eight of the histories reviewed by Aird and in fifteen of our collection. It is interesting that achlorhydria was present in only four cases, and that in most of the cases the acids were normal or excessive. This finding has been offered as further evidence that carcinomas which arise in the presence of normal acids are ulcer-carcinomas and arise in pre-existent peptic ulcers. It can also be reasoned that a penetrating peptic ulcer can develop in a carcinoma. The break in the mucosa initiated by a new growth exposes the underlying tissue to the digestive action of the gastric secretions which may rapidly penetrate the wall and be responsible for the perforation. Irrespective of this academic question certain facts stand out. The patients with normal and high acids had long histories of dyspepsia, usually typical of ulcer, and upon perforation presented a clinical picture and pursued a course identical with that of perforated peptic ulcer.

Too little data are available to determine definitely the influence of achlorhydria. Such as there is, suggests that carcinoma in the absence of acid perforate as a result of necrosis rather than ulceration and the perforation is likely to be relatively painless and silent, or if sudden and painful, then rapidly fatal from septic peritonitis.

The two important problems relating to perforated carcinomas of the stomach are first, recognition of the carcinoma by routine biopsy and second, operative management. In approximately 50 per cent of cases a hopelessly incurable situation exists because of advanced peritonitis from septic soiling or delayed intervention or because the tumor is not resectable.

There is no question about the desirability of resection of resectable lesions—only a question of when it should be done. In an endeavor to answer this question the results of the various methods of management used in 115 operations in cases of the collected series are shown in Table I.

TABLE I

	RECOVERED	HOSPITAL DEATHS
Drainage alone	3	15
Simple closure (with or without drainage)	9	34
Local excision with simple closure	2	4
Simple closure and gastroenterostomy	7	16
Simple closure and jejunostomy	0	2
Jejunostomy alone	0	2
Primary simple closure and resection at subsequent operation		
Planned	1	1
Corrected*	2	2
Primary resection	13	2
Total	37	78

*Perforation at primary operation was misdiagnosed peptic ulcer and proved malignant by subsequent developments and studies.

A mortality of 80 per cent following simple closure of perforations is understandable. Several reports contained remarks about the difficulty in obtaining satisfactory closure. Because sutures cut through the rigid and friable carcinomatous tissue, a few surgeons had to resort to suturing a patch of omentum over the perforation. In most instances post-mortem examination showed failure of healing with continued or subsequent leakage. This very considerable and inescapable hazard vitiates the theoretical advantage of the staged method of management; that is, emergency closure of the perforation by suture followed some days or weeks later by gastrectomy. In our collected series, 6 of the patients who recovered following primary closure were subsequently subjected to gastrectomy. Only 3 of them, or 50 per cent, survived. In 2 cases the two-staged procedure had been planned at the initial operation; in 4 the perforation had been closed in each instance upon the assumption that it was a perforated ulcer. After subsequent events and studies had proved these patients to have perforated carcinomas, resection was done. Furthermore, it is apparent from the histories that several additional patients who failed to survive simple closure had resectable lesions which probably would have been resected at a second operation.

For comparison with this record of two-stage management there are available in the literature the records of 15 primary resections, with the seemingly incredible result of only 2 fatalities, a mortality of only 13 per cent.

In the series of cases collected by Aird there were 7 primary gastrectomies with no mortality. McNealy and Hedin reported 7 primary gastrectomies with 2 fatalities in a group of cases collected by them from the records of Cook County and Wesley Memorial Hospitals. Both authors expressed the opinion that the superior results from primary gastric resection should be accepted with certain reservations. They believe that it was reasonable to assume that patients subjected to gastrectomy were in the most favorable general condition and that there is probably a tendency to report solitary successes and for the failures to be absent from the literature. These intangible factors in the available data make a comparative estimate of the merits of the single- and two-stage operation impossible. However, it is my opinion that the hazard of a gastrectomy in the presence of a diffusely soiled peritoneal cavity has been grossly overestimated and the hazard of leakage from a sutured perforation in carcinomatous tissue much underestimated.

CASE 1.—J. S., a Negro waiter, 49 years of age, was admitted to the hospital department of the Union Pacific Railroad, Dec. 6, 1942, writhing with generalized abdominal pain. The onset had been sudden, four hours previously, with severe pain in the epigastrium which diffused rapidly throughout the abdomen and then to the top of both shoulders. He had vomited a small amount of clear fluid soon after the onset.

He related that for one year he had had burning and gaseous distress in the epigastrium, which came on two or three hours after meals and during the night, and that this distress was relieved by the ingestion of food or alkalies.

On his admission, three months previously, a diagnosis of duodenal ulcer had been made upon the basis of a persistent deformity of the duodenal cap, with a 10 per cent five-hour gastric retention, normal free and total acids, and the repeated presence of occult blood in the stools. After ten days' hospitalization on an ulcer regime he was totally, if only temporarily, relieved of symptoms.

The patient presented the classical signs of perforated ulcer, with the confirmatory roentgenographic evidence of free gas in the peritoneal cavity.

Operation was performed one hour after admission and five hours after onset of symptoms. When the peritoneal cavity was opened there was an escape of much gas and "dishwater" fluid. The latter seeped from a diffused distribution throughout the abdomen. The stomach, delivered into the wound, presented a small perforation on the superior aspect of the anterior wall approximately three inches proximal to the pylorus. Palpation revealed that the perforation was located in the center of a crater the size of a silver dollar which had a wide, hard elevated border confluent inferiorly with a similar border surrounding a smaller crater. Except for a few glands in question along the lesser curvature, there was no evidence of metastases. A small piece of tissue was excised from the edge of the perforation and examined in frozen section. The pathologist returned a report of adenocarcinoma.

Confronted with a situation regarding which I had no knowledge, I reasoned that healing of a perforation through carcinomatous tissue would be unlikely and since the lesion appeared to be a very favorable one for resection, an immediate subtotal gastrectomy, even in the presence of a soiled peritoneal cavity, seemed a justifiable and logical procedure. Consequently, resection of the Polya-Balfour type was done. Five grams of sulfanilamide crystals were distributed over the peritoneal surfaces, and the peritoneal cavity was closed without drainage. The wound in the abdominal wall was washed copiously with saline solution, dusted with sulfanilamide crystals, and drained.

The pathologist's report of the specimen was as follows: A large segment of stomach with attached mesentery. Eight centimeters from the pylorus there was a crater which measured 4½ cm. in diameter, with a central perforation the size of the head of a match. Adjacent was a smaller crater measuring 1½ cm. in diameter. Both were surrounded by induration which disrupted the normal mucosal pattern. On the serosal surface there were thickening and fibrinous changes over the area of perforation. No glands containing carcinoma were found in the attached tissue. Sections from the borders of the crater show well-formed glands with well-differentiated cells containing hyperchromatic nuclei. There was occasional mitosis, and invasion of the muscularis. Diagnosis: Perforated ulcerating adenocarcinoma of the stomach, Grade II, with no metastases to nodes of the attached mesentery.

The immediate convalescence was entirely uneventful. Ten days following dismissal from the hospital the patient developed a fever and a pleuritic type of pain at the base of the right chest. He was readmitted to the hospital and a small right subphrenic abscess drained.

He returned to work in four months and remained free of symptoms and apparently well, for one year and four months. He then developed an ascites with general carcinomatosis peritonea and died twenty months following operation.

In this case, there was the advantage of a record of complete gastrointestinal studies three months prior to perforation. The normal acidity of the gastric contents which soiled the peritoneal cavity was probably

an important factor in preventing a bacterial peritonitis. The typical ulcer history of one year's duration, in addition to normal acidity, raises the question whether the carcinoma was the sole and primary lesion or had its origin in a pre-existent benign ulcer.

PERFORATED PEPTIC ULCERS

Associated Recent or Simultaneous Hemorrhage.—An impression of the incidence of hemorrhage in association with perforation can be gained from the report of McNealy and Howser. In their series of 700 cases there were 21 patients who had gross hemorrhages with the onset of perforation. In DeBakey's series of 2,525 cases, there were 155, or 6.1 per cent, incidences of hemorrhage. Simultaneous hemorrhages occurred in 16, or 10.5 per cent, of the 152 gastric perforations reviewed by Winters and Egan. Harrison and Cooper reported 8 instances out of a series of 57 cases. Five of these patients died immediately following the operation of simple closure and one subsequently had a fatal hemorrhage. As judged from this and similar references, hemorrhage contributes enormously to mortality and it would appear that simple closure of the perforation is inadequate because too often it fails to control the hemorrhage. Post-mortem examination has shown that these patients often have multiple ulcers and that hemorrhage usually arises from the nonperforated one, often in the case of the duodenum, from the posterior of the two kissing ulcers. Harrison and Cooper were able to demonstrate an open artery in one such case. I believe that gastric resection which removes the entire ulcer-bearing area and all sources of hemorrhage is indicated in these cases. A single experience of this kind is presented in Case 2.

Case 2.—D. A., 48 years of age had had recurring ulcer symptoms for four years. He was admitted to the hospital five hours following perforation. For ten days prior to perforation he had had pain intractable to ulcer regime and four days previously had noted a tarry stool. At the onset of perforation he had vomited considerable coffee-ground material, felt faint, and had a large tarry stool; he later twice vomited blood. Examination revealed boardlike rigidity of the abdomen and acute tenderness throughout, with roentgenologic evidence of free air in the peritoneal cavity. The blood pressure of 100/70, the pulse of 110, and the marked pallor all suggested active hemorrhage. An immediate exploration was done and 1,000 c.c. of bank blood administered through needles in both arms simultaneously. There was diffuse but minor soiling of the peritoneum with grossly bloody fluid. On the lesser curvature about two inches distal to the cardio-esophageal junction, there was a small perforation which at the moment seemed to be sealed by a bloody fibrinous clot which was adherent to the anterior surface of the stomach about the perforation. A second ulcer slightly distal and on the posterior wall could be palpated. A biopsy was taken from the margin of the perforation for frozen section and this proved to be only inflammatory, with no evidence of carcinoma. The stomach contained a considerable quantity of blood. An antecolic Hofmeister type of resection was performed, 8 Gm. of sulfanilamide crystals were placed in the site of operation and abdominal wall, and the abdomen was closed without drainage. Following an uneventful convalescence, the patient

left the hospital in fourteen days. The gross specimen showed kissing ulcers high on the lesser curvature, the anterior one perforated, the posterior one penetrated to the serosa. The site of hemorrhage could not be definitely determined.

REPEATED OR RECURRENT ACUTE PERFORATION

Recurrent perforations are uncommon. In DeBakey's collection of 6,538 cases there were 74, or 1.1 per cent, reperforations. Davenport and Henry have each reported a case in which five reperforations occurred, and there are several recorded cases of three and four recurrent perforations. Patients in this category develop very acute ulcers which perforate with little or no warning. Because the records indicate that many of them have an ulcer diathesis and eventually require resection, there would seem to be adequate indication for an emergency resection at the time of the second perforation if performed under the favorable circumstances of early operation on a patient in good condition.

CASE 3.—G. B., 38 years of age, had had two previous perforations four years and ten months before, both treated by simple closure. He was admitted eight hours after the present or third perforation, and stated that for ten days he had vomited daily. Typical findings were presented, including roentgen evidence of free air in the abdomen.

At operation there was diffuse soiling but only a moderate amount of fluid. There was an astounding absence of adhesions from the previous perforations and operations. The stomach appeared to be dilated, and at the pylorus on the anterior wall there was a small perforation in the center of an indurated area which involved the entire circumference of the contiguous duodenum. The sites of previous perforations could not be identified.

Because the ulcer appeared to be acute, because it was the third one to perforate, and because there was evidence of pyloric obstruction, a subtotal anterior Polya gastrectomy was done. All fluid possible was aspirated from the peritoneal cavity, 10 Gm. of sulfanilamide crystals were placed around the site of operation and in the abdominal wall, and closure was effected with drainage of the wall only.

Convalescence was uncomplicated and the patient left the hospital in eleven days.

Examination of the specimen revealed, in addition to the perforated one, a penetrating kissing ulcer on the posterior wall and a small shallow prepyloric posterior ulcer.

PERFORATION ASSOCIATED WITH PYLORIC OBSTRUCTION

Perforated Jejunal Stoma Ulcer.—Only occasionally is the combination of fixed or permanent obstruction at the pylorus and acute free perforation encountered. Often the edema and induration about a perforated duodenal ulcer so narrows the lumen that many surgeons early in their experience have had much anxiety about restoration of patency following closure of the perforation, and not infrequently have been led to supplement closure with gastroenterostomy. Fortunately, the inflammatory process rapidly subsides and an adequate lumen is restored in all but a few cases. These may be designated as fixed in contradistinction to the apparent obstructions due to swelling. Fixed obstruction is the result of scarring incident to long-standing ulceration, often multiple ulcers. In these cases a by-pass must be provided and for this purpose gastroenterostomy is a poor second choice to resection.

In the patient whose history follows (Case 4), a gastroenterostomy had been performed and had proved inadequate. Not only had it failed to cure or prevent subsequent ulceration in the duodenum but also it had added a new source of trouble, an ulcer at the stoma which had perforated. This complication of ulceration in or about the stoma, or more often in the efferent loop of the jejunum, occurs not infrequently following gastrojejunostomy but only occasionally following subtotal gastrectomy. The incidence following gastrojejunostomy has been estimated at figures varying from 1.4 (Walton) to 34.0 (Lewisohn) per cent. There is also a wide difference of opinion regarding the frequency with which these ulcers perforate into the free peritoneal cavity. Liebein reported 30 perforations in 129 cases, an incidence of 23 per cent. Toland and Thompson who collected 93 cases, all that they were able to find in a search of the literature to 1936, estimated the incidence of perforation at less than 1 per cent.

The primary objective in the management of this emergency, as in the case of perforated gastric and duodenal ulcers, is the immediate survival and recovery. It would seem that simple closure is the simplest and therefore the safest method of accomplishing this objective. Curiously enough, this is not the case as judged by the evidence available in the literature. In the series collected by Toland and Thompson, simple suture of the perforation was carried out in 51 cases with 9 deaths, a mortality of 17.6 per cent. In 17 cases, the emergency was handled by subtotal gastrectomy with only 1 death, a mortality of only 5.8 per cent. Disconnection of the gastrojejunostomy restoring the normal sequential relation of the stomach and intestine was utilized in 4 cases with no deaths. Gastroenterostomy alone or in combination with other procedures was used in 7 cases with 2 deaths, a mortality of 28.5 per cent.

In only 1 case in this entire series was there reported the association of a perforated jejunal ulcer and fixed pyloric obstruction. As in my case, a subtotal gastric resection was done.

CASE 4.—J. J., 41 years of age, had had a posterior gastrojejunostomy two years previously for a duodenal ulcer which was intractable to therapy. He had been free of symptoms for one and one-half years, but during the past eight months symptoms had recurred and relief was obtained only with constant adherence to an ulcer regime. For two weeks there had been much pain at the umbilicus and in the back (about the first lumbar vertebra). It occasionally radiated into both testicles.

Four hours before admission the patient was seized with severe generalized abdominal pain and vomited only a small amount of bile-stained fluid. The clinical picture was typical of perforation. The blood studies revealed only a slight anemia, hemoglobin 12.5 Gm. and red blood count 4,000,500.

At operation, which was done immediately, diffuse and copious soiling and free gas were encountered, as were extensive adhesions between the abdominal wall, the omentum and stomach, and between the duodenum and liver. After retracting the transverse colon upward, the point of leakage was traced to a small perforation on the right anterior aspect of the gastrojejunal stoma. After adhesions were

freed, an ulcer was found on the superior aspect of the first portion of the duodenum and an apparent scar on the anterior aspect. The lumen appeared to be almost occluded at this point.

The anastomosis was taken down, the stomach subtotally resected, and a posterior full stoma anastomosis done.

As in the previous cases, sulfanilamide crystals were used and drainage was instituted for the abdominal wall only.

The patient had a very stormy convalescence as a result of atelectasis of the right lower lobe and what appeared to be peritonitis, followed on the eighth day by total wound disruption. However, he left the hospital in good general condition on the twenty-second day.

VERY EARLY PERFORATION IN THE VERY YOUNG

Under this heading are included the cases with insignificant soiling. The choice between resection and simple closure in this group is debatable and is influenced by individual factors such as the temperament and occupation of the patient and the inclinations and ability of the surgeon. In the hands of skilled surgeons, resection will be attended by little or no more primary mortality than simple closure, and the percentage of permanent cures will be much larger. The majority of individuals under 35 years of age do not remain free of ulcer symptoms following simple closure and in many instances the recurrent ulceration necessitates surgical intervention. Case 5 represents this group.

CASE 5.—D. B., 26 years of age, was admitted Dec. 8, 1943, two hours following perforation. He had had recurring ulcer symptoms for two years, which had always responded to ulcer therapy until the present episode which had lasted five days.

Immediately after eating his evening meal he was seized suddenly with severe general abdominal pain, worse in the epigastrium. Upon admission he presented the typical clinical picture of a patient with perforated ulcer, including radiographic evidence of free air in the peritoneal cavity.

At operation, which was performed immediately, there was diffuse soiling but only a moderate amount of fluid. On the anterior surface of the duodenum, just distal to the pylorus, there was a small perforation in the center of a wide area of induration. The duodenum appeared to be fixed posteriorly and there seemed to be palpable evidence of an ulcer on the posterior wall. In view of the two-year ulcer history, the athletic constitution of a man of 26 years, and a perforation of only two hours' duration, resection was performed as the procedure of choice. An anterior Polya type of operation was done; 8 Gm. of sulfanilamide crystals were placed in the abdomen and abdominal wall and a cigarette drain inserted to the peritoneum.

Convalescence was uneventful and the patient left the hospital twelve days after the operation.

CONCLUSIONS

1. From available statistical data it appears that subtotal gastric resection can be performed in the presence of diffuse soiling of the peritoneal cavity within twelve hours after the perforation of ulcerating lesions of the stomach, duodenum, and jejunum in good risk cases with a lower mortality than that obtained by simple closure with sutures. With few exceptions, resection results in permanent cure in contrast

to the high incidence of recurrent ulceration following simple closure of the perforations. This advantage is a justifiable consideration in the choice of operation by surgeons skilled in radical gastric surgery.

2. However, simple closure of perforations with sutures can be performed satisfactorily by surgeons with comparatively little experience, and subsequent radical therapy can be carried out under more favorable circumstances in the group of patients who develop recurrent ulcers with complications necessitating operation. For these reasons the immediate interests of most will be best served by simple closure.

3. There are, however, a few cases in which emergency primary resection is the operation of choice or has advantages that make it almost imperative. In the latter group are: (a) perforated resectable carcinomas of the stomach, (b) perforated peptic ulcers with simultaneous hemorrhage, and (c) perforated peptic ulcers with fixed pyloric obstruction. (Gastroenterostomy is an inferior alternative.)

In the other group the choice between simple closure and resection is debatable but resection is a justifiable preference in experienced hands because it offers much more assurance of permanent cure. Constituting this group are: (a) perforated peptic ulcers with insignificant or only local soiling (local soiling occurs in all elective resections except those performed by the so-called closed aseptic technique), (b) recurrent perforations, and (c) very early perforations in the very young patients who are otherwise in excellent health. In this group the incidence of recurrence is very high, the risk from resection very small, and the need for a permanent cure and good health very important.

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SURVIVAL AFTER GASTRIC RESECTION IN CARCINOMA OF THE STOMACH

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THIS paper is presented as a review of the cases of carcinoma of the stomach in which the patients have been operated upon at the Highland-Alameda County Hospital. It is not the intention to study here all the patients with possible malignancies who have been admitted, treated or untreated, carcinoma diagnosed or suspected, only to look in retrospect at those patients who were considered operable after the abdomen was opened, had a pathologic diagnosis of the lesion, and have been duly followed through their succeeding years. Only by taking as a control group, cases so completed, can accurate and dependable information be given as to the results obtained at this hospital over this period of study.

In studying past cases of this type there could be an endless number of factors considered which, rightly, are weighed before the surgeon takes the patient to the operating room. And each of these factors, howsoever seemingly insignificant, could well have a major bearing on the cure or convalescence, but should each factor be discussed individually this would be an endless paper. So it has been assumed that if these patients have gone through major surgery, convalesced, and been closely watched over a period of years that such factors as their degree of anemia, weight loss, location of lesion, achlorhydria, and size as shown by film will not bear the significance to the patients in measuring the results obtained as the one which is to be taken into consideration—that of their longevity or survival rate.

From 1930 to 1944, an interval of fourteen years, exploratory laparotomies, contemplating palliative or curative procedures were performed on 463 patients with carcinoma of the stomach. From this total, there were 141 considered operable after the abdomen was opened. The follow-up clinic was able to carry through with a total of 96 cases, each having preoperative clinical and laboratory diagnosis of gastric malignancy, each confirmed by a pathologic diagnosis of the lesion removed, and each having autopsy at the time of death.

In studying this controll group of 96 on a follow-up basis, they have been divided into: (1) Operative deaths, (2) postoperative complications, (3) death under three years, (4) death under five years, (5) death under eight years, and (6) survival over eight years.

There were two patients who died during surgery; one death was attributed to hemorrhage and one was an anesthetic death, making

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mortality during operation, 2.03 per cent. Of the fatal postoperative complications, three were latent hemorrhages, one subdiaphragmatic abscess, two generalized peritonitis, three postoperative pneumonias, or a total of nine, making 9.37 per cent mortality. Combining these two there was an operative risk of 11.39 per cent.

Of the 23 patients who did not survive over three years, not including the operative deaths, all had an extension of the lesion with metastasis. This group comprises 23.95 per cent of the original, which when added to those succumbing to surgery makes 35.34 per cent who failed to live over three years. For a little divergence and as a matter of comparison, it is interesting to note here that there were 28 patients seen in the hospital on whom a clinical and laboratory diagnosis of operable malignancy of the stomach had been made and who later came to autopsy at this institution. Of these patients, 100 per cent failed to live over three years. All 28 had refused surgery.

Still carrying this further, 36 patients, or 37.59 per cent, died in the three- to five-year interval, two from accidents, one from coronary disease, and one from cerebral accident. These five had no evidence of recurrence of their original lesion.

If the criteria of cure are to be based on a five-year curve, then there were 26 patients or 27.06 per cent, who survived. These resections appeared excellent, but 8 of these patients did not live over the eight-year period, 6 expiring from the recurrence of their original lesion and 2 from other pathologic conditions.

The next group narrows considerably. In this, there are a total of 18 patients who were alive and well for over eight years and symptom-free as far as their previous condition was concerned, a survival rate of 18.75 per cent over eight years. How do these results compare with those reported elsewhere? In 1942, the Mayo Clinic published an extensive study of carcinomas and other malignant lesions of the stomach, a series of 1,968 patients. In this publication, they reported a 28.9 per cent survival over five years, and 20.4 per cent over ten years, as against 27.06 per cent and 18.75 per cent for this institution.

To summarize, over an interval of fourteen years at this hospital, there were 141 patients with an established diagnosis of carcinoma of the stomach on whom gastric resection was performed. After five years, 27.06 per cent were alive, and 18.75 per cent had a survival rate over eight years. In contrast, there was a group of 28 in whom the diagnosis of gastric cancer had been established. This group of patients refused surgery and the mortality at the end of three years was 100 per cent.

THE CREATION OF A GASTRIC POUCH FOLLOWING TOTAL GASTRECTOMY

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THERE are still many problems confronting us in total gastrectomy, not only in the operative procedure but in the postoperative care as well. A problem which has attracted our attention recently perhaps has concerned others dealing with this type of surgery. That is, the inability of some patients following total gastrectomy and with an esophagojejunostomy to handle even small feedings without a feeling of fullness and discomfort. This can be a distressing symptom to the patient and of great concern to the surgeon since it presents a real problem as far as nutrient food intake is concerned.

Jordan,[1] of the Lahey Clinic, has reported a case of a woman who, after total gastrectomy and esophagojejunostomy, vomited and complained of substernal pain. This proved to be due to contracture of the anastomosis. These symptoms were relieved after dilatation of the anastomosis. I am not discussing the distress due to this complication but that due to the lack of physical space in the jejunum to handle a moderate-sized meal.

I would like to report here a case of similar symptoms, but one not due to stenosis or spasm of the esophagojejunal anastomosis. The symptoms were relieved by a secondary operation creating a pouch out of the afferent and efferent loops of jejunum.

CASE REPORT

A 57-year-old man was operated upon May 14, 1944, and a large peptic ulcer was found on the lesser curvature of the stomach involving the lower 1.3 cm. of the esophagus (Fig. 1). Because of the induration of the rest of the stomach and an associated gastritis it was decided that a total gastrectomy was the only answer to the problem.

A total gastrectomy was done by first ligating the right and left gastric arteries and freeing them from the lesser curvature. The right and left gastric epiploic vessels were then ligated and their branches freed with the gastrocolic omentum from the greater curvature. The pylorus and duodenum were then mobilized. At this point the first portion of the duodenum was transected between clamps (Fig. 2). The distal stoma of the duodenum was closed with continuous chromic catgut No. 0 suture. This stump was then inverted with a linen purse-string suture, after which a row of interrupted sutures of linen were placed as seroserosal sutures. The closure of the duodenal stump is of paramount importance and cannot be stressed with too much emphasis (Fig. 3).

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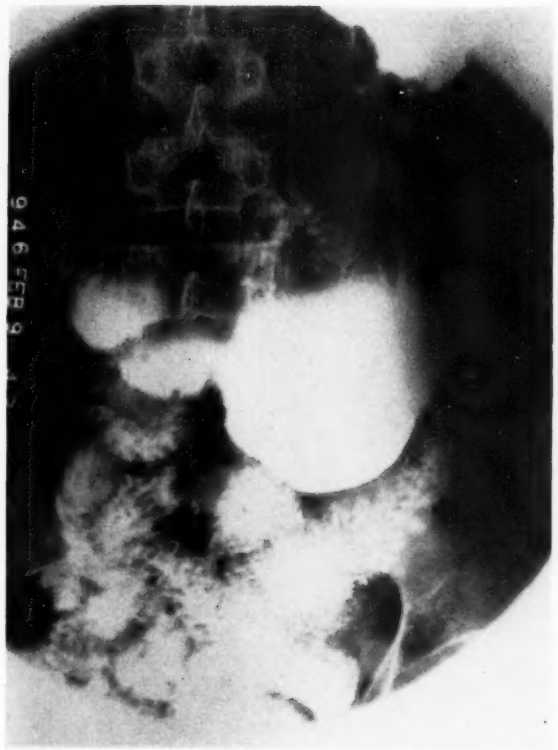
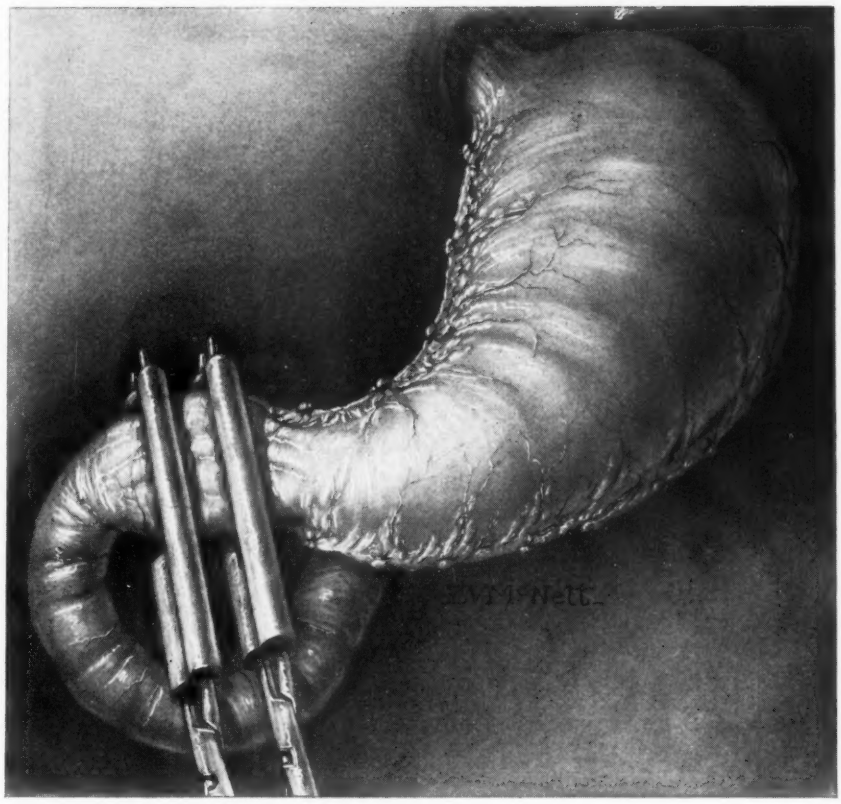


Fig. 1.—Preoperative x-ray picture showing a small, penetrating ulcer high on the lesser curvature of the stomach.

Fig. 2.—Right and left gastric vessels and right and left gastric epiploic vessels have been ligated and separated. This diagrammatic sketch shows clamps in place for transection of duodenum.



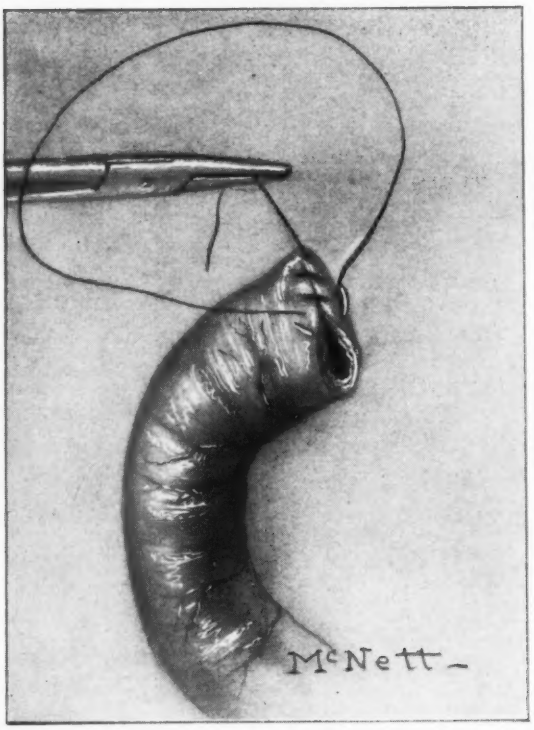


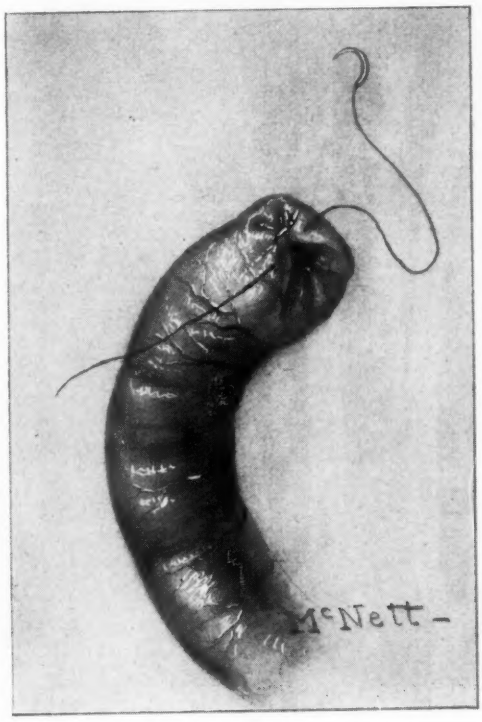
Fig. 3*A*.—Continuous suture for closure of duodenal stump.

Fig. 3*B*.—Stump closed with continuous suture, and purse-string suture being put in place.

C.

D.

Fig. 3*C*.—Burying of stump with purse-string suture.
Fig. 3*D*.—Purse-string suture tied tightly, burying stump.



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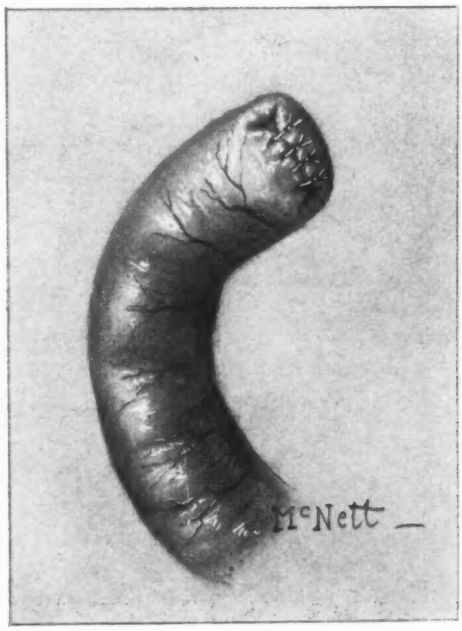


F.

Fig. 3*E*.—Interrupted linen seroserosal sutures are being placed.
Fig. 3*F*.—Completion of stump closure.

The reflection of the diaphragmatic peritoneum was then freed where it overlies the esophagus. May I point out here that the freeing of this peritoneal reflection allowed excellent mobilization of the esophagus. The anastomosis of the esophagus to the jejunum was done after the method of Lahey,[2] with a long jejunal loop brought up anterior to the transverse colon. It was felt wise here to amputate the great omentum to prevent drag on the afferent jejunal loop.

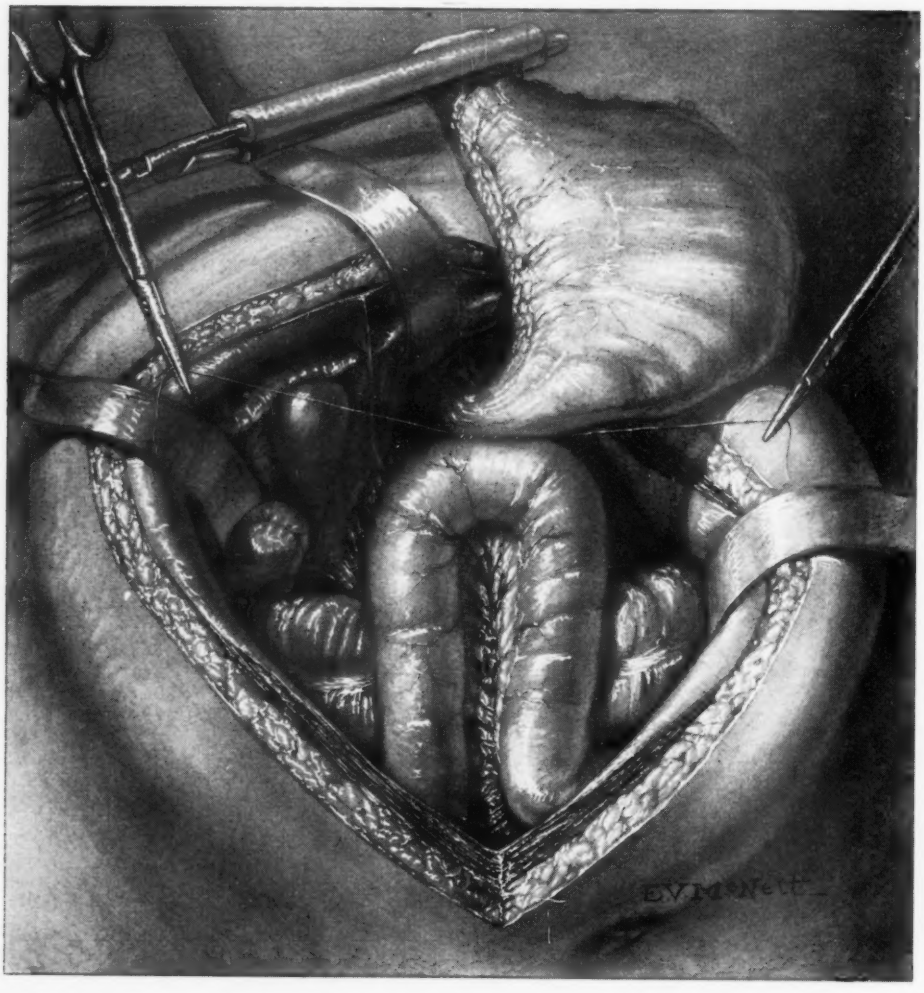


Fig. 4.—The posterior suture line of the esophagus to the jejunum is in place with stomach retracted upward.

In doing the anastomosis, it simplified the procedure to use the stomach to drag the esophagus downward and forward. A line of sutures was placed at the posterior suture line attaching the jejunum to the posterior aspect of the esophagus (Fig. 4). After this row of sutures were placed, the posterior wall of the esophagus just distal to the suture line was incised as was the jejunum. A second suture line (continuous No. 0 chromic catgut) was placed through the posterior layer of jejunum and the posterior wall of the esophagus (Fig. 5). After this the stomach and portion of the esophagus were removed by cutting through the anterior wall of the esophagus. The continuous suture was carried

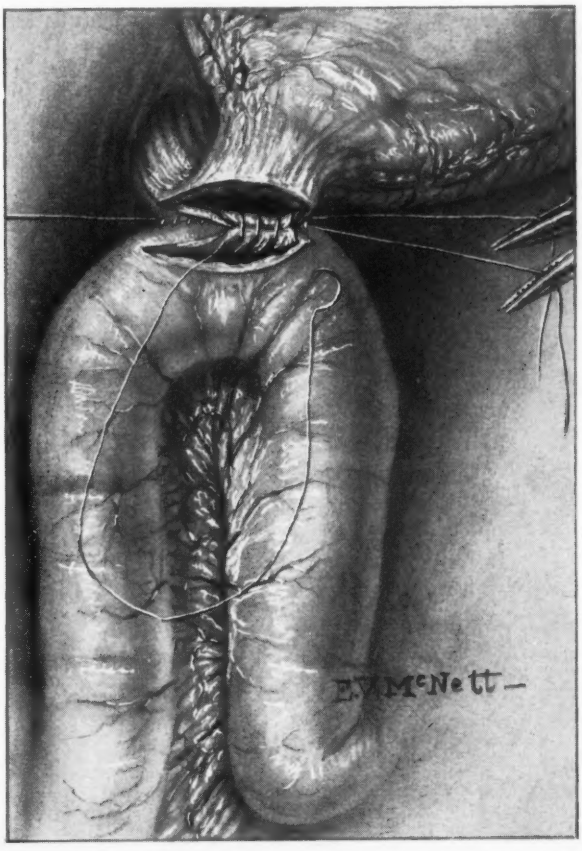
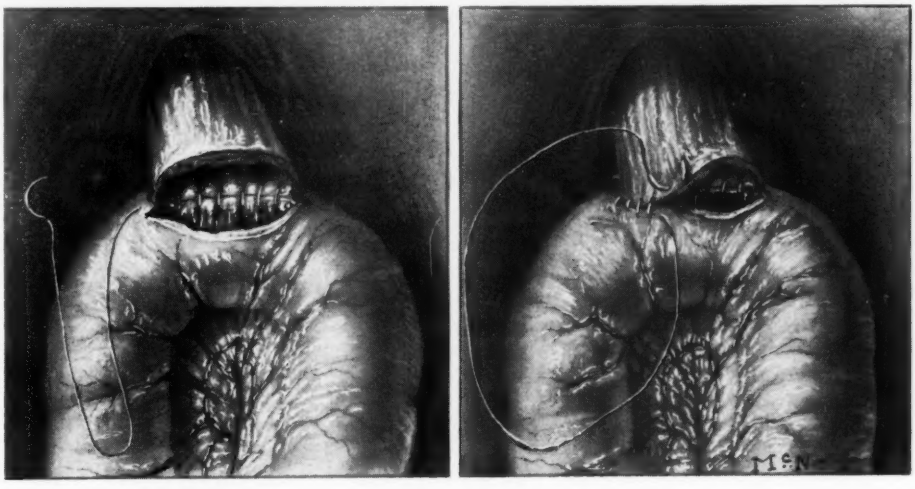
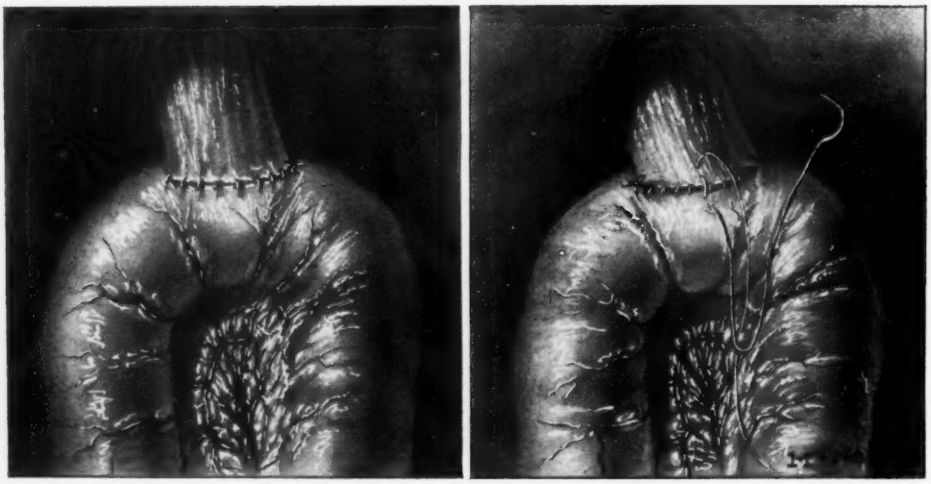


Fig. 5.—The beginning of the anastomosis to form the posterior wall of the esophagojejunostomy. Stomach is still being used for traction of the esophagus.



A. *B.*

Fig. 6.—*A*, Completion of posterior closure of esophagojejunal anastomosis. *B*, The beginning closure of anterior portion of anastomosis with the Lembert suture.



A. *B.*

Fig. 7.—*A*, Completion of anterior closure of esophagojejunal anastomosis. *B*, One of four reinforcing sutures being put in place in the anastomosis.

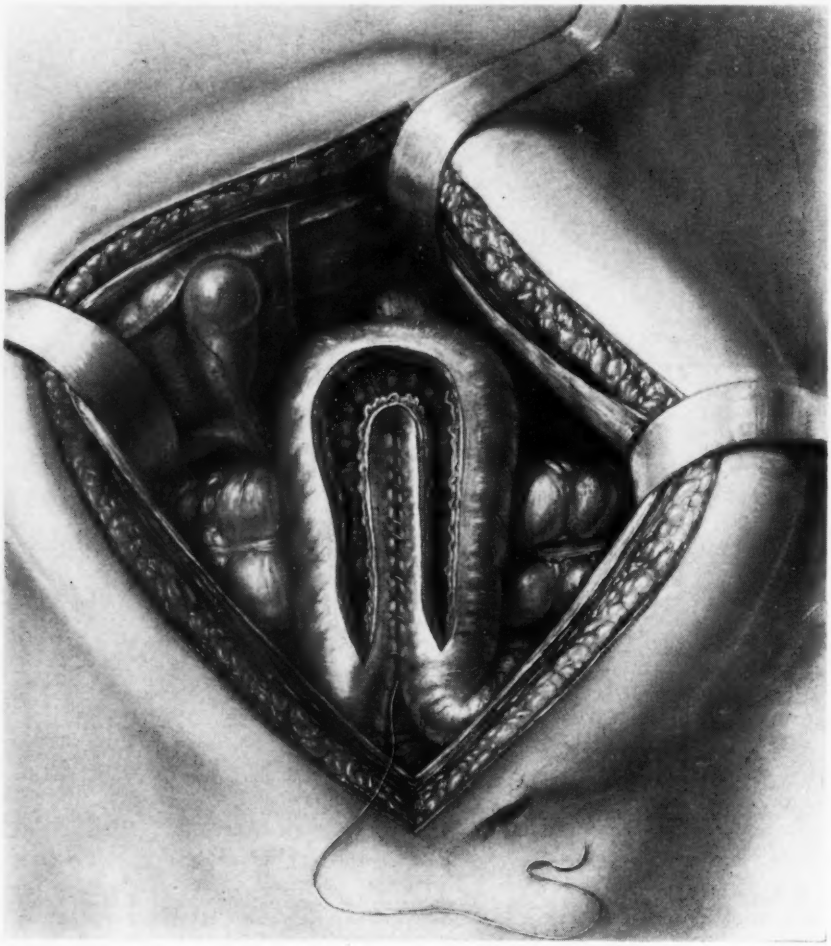


Fig. 8.—Seroserosal sutures between the afferent and efferent loops of jejunum in place and incision of the jejunum completed.

anteriorly to close the anterior wall of the jejunal opening to the anterior wall of the esophagus. Following this a second row of sutures was used to reinforce this line. Four interrupted sutures were placed at equal points around the anastomosis, through the muscular wall of the esophagus to the serosa of the jejunum for reinforcing the anastomosis and taking the drag off the anastomotic line (Figs. 6 and 7). Still another row of sutures was placed from the serosa of the jejunum to the diaphragmatic reflection of the peritoneum which was originally freed. A Jutte tube was passed down the esophagus and into the efferent loop of jejunum for a distance of about 8 cm. This was to be used for postoperative feeding. The abdomen was then closed in layers without drainage.

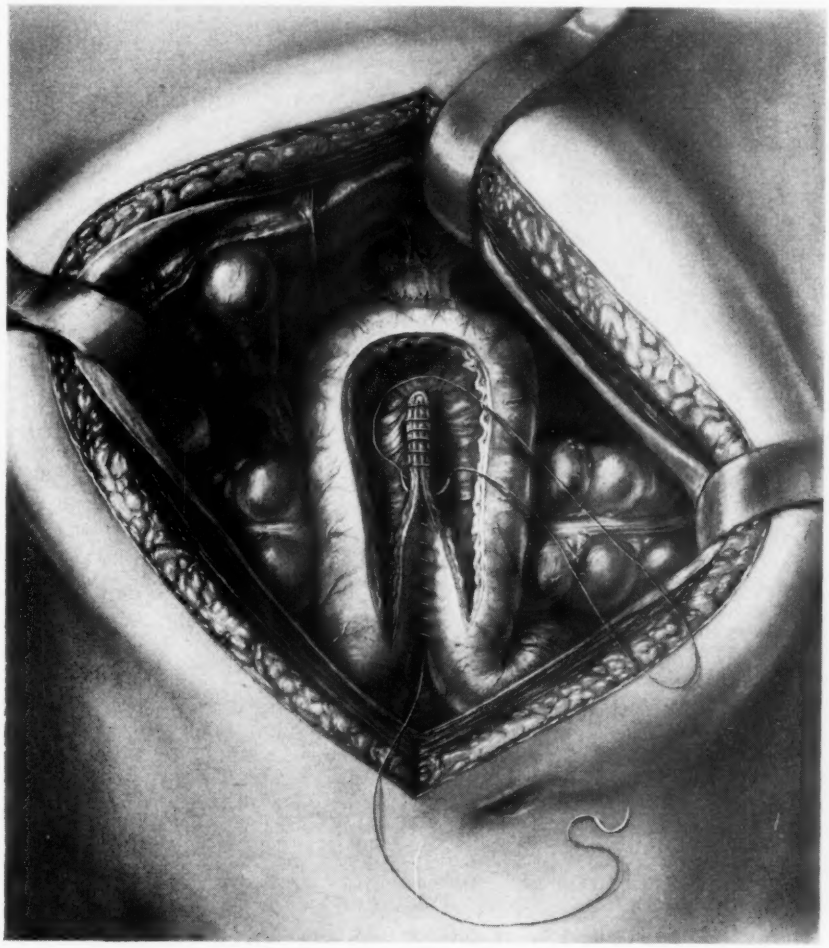


Fig. 9.—The beginning of the suture through the six layers of the two loops of jejunum forming the posterior wall of the pouch.

The feedings were carried on at 30 Gtt. per minute. These feedings alternated 500 c.c. of Karo syrup in water and 500 c.c. of predigested beef.* In twenty-four hours 1,000 c.c. of each mixture was given. This totaled 1,770 calories. Liquids were started by mouth and the feeding tube removed on the sixth day.

*Select one-half pound of beef. Remove everything that is not clear meat. Chop fine. Put in pint fruit jar and add one cup of cold water and five drops of dilute hydrochloric acid. Stir and set in refrigerator or any cool place to digest for two hours. Strain, season, and serve in drip bottle.

Soft diet was started on the seventeenth postoperative day on a six-feeding basis. The patient complained of fullness and distress after eating. A barium x-ray picture was made which showed the anastomosis to be normal sized and functioning. The complaint and symptoms of distress continued, and on the twenty-eighth postoperative day it was decided to reoperate and make a pouch out of the afferent and efferent loops of jejunum.

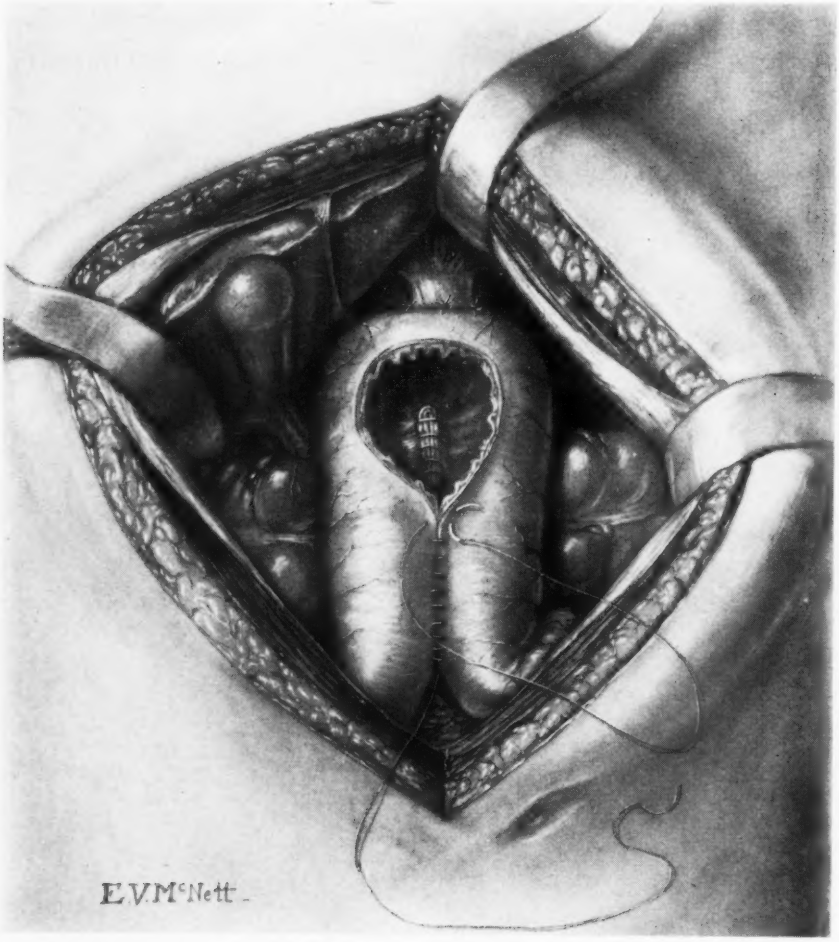


Fig. 10.—The start of the closure of the anterior wall of the pouch by a Lembert suture. The original seroserosal suture is seen in the lower portion.

I reoperated upon the patient through the same upper right rectus incision. The peritoneum was opened and the anastomosis was examined. It was found to be patulous and one finger in diameter. The afferent and efferent loops of jejunum were brought next to each other. Beginning at the upper angle formed by these two loops, a seroserosal suture was started about 1.5 cm. from the mesenteric attachment. This suture was carried down, suturing the afferent and efferent loops together for a distance of 8 cm. (Fig. 8). An incision was then made through all three layers of the afferent loop near the seroserosal suture line. This incision was carried upward on the afferent loop to the angle of the jejunum near the anastomosis, across this angle and down the efferent loop to the lower end of the seroserosal suture (Fig. 9). The medial walls of the afferent and

efferent loops of jejunum were then sutured together, starting at the upper angle with a continuous chromic No. 0 catgut. This suture completed the posterior wall of the pouch. When the lower angle of the incision was reached the suture was continued as a Lembert suture, bringing together the lateral wall of the incision in the afferent loop to the lateral wall of the incision in the efferent loop (Fig. 10). When this suture was completed the original seroserosal suture was continued up the anterior wall overlying the Lembert suture line and completing the closure of the anterior wall of the pouch (Fig. 11). The abdomen was then closed in layers.

Fig. 11.—The completion of the seroserosal suture up the anterior wall of the two anastomosed loops of jejunum, completing the formation of the pouch.

The postoperative course of the patient was uneventful. Tube feedings were started through a Jutte tube which had been passed down the esophagus, through the pouch, and down the efferent loop of jejunum. Mouth feedings of liquids were started on the third postoperative day, when the feeding tube was removed. Soft diet was started on the eighth postoperative day and light diet on the fourteenth day. The patient did well and had no complaints. X-ray on the seventeenth day after the pouch was made showed it to be working fine. The patient was discharged the following day (Fig. 12).

A fractional test meal of the pouch contents showed no free hydrochloric acid. The total acidity was 22-24. Serum protein varied from 5.75 to 6.29 per cent.

Six months after operation the patient's hemoglobin was 91 per cent, red blood cells 5,220,000, serum protein 6.12, and blood chlorides 444 mg. per cent. A fractional test meal showed no free hydrochloric acid, no free lactic acid, and a total acidity of 2.5. The patient's only complaint was a restricted diet. He had gained fifteen pounds since operation and was on a full diet of three meals per day. Complete mastication of food was stressed as the most important thing to future health. At that time he was working in a war plant doing a full forty-eight hour a week job.

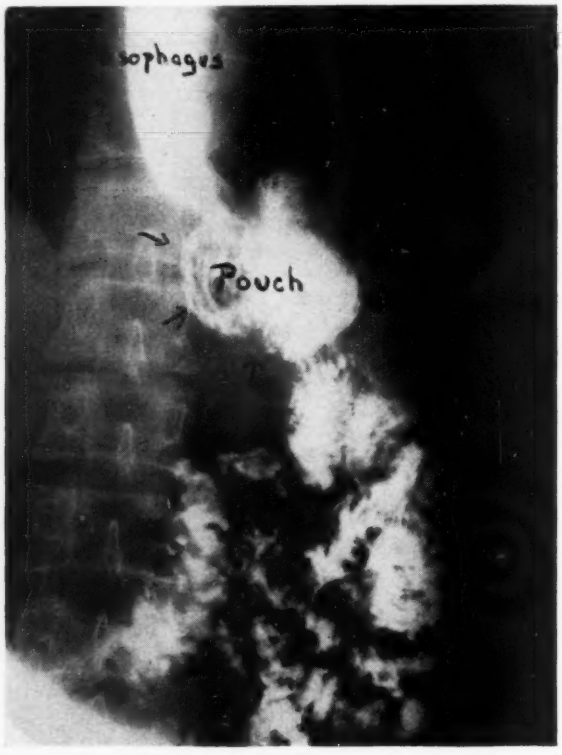


Fig. 12.—X-ray of patient before discharge, showing pouch filled with barium.

I have since operated upon a 66-year-old woman for carcinoma of the fundus of the stomach. In this case I did a total gastrectomy after the Lahey method and at the same time made a pouch of the afferent and efferent loops, all at one stage. This patient made an excellent recovery and never had a complaint of distress or fullness on eating.

The technique for this pouch operation was somewhat similar in manner to the Finney[3] gastroduodenostomy except that instead of using stomach and duodenum the anastomosis was made between the two loops of jejunum.

Another great aid in gastric surgery is the recommendation by Allen and Donaldson[4] of the use of jejunostomy for decompression and feeding. In the total gastrectomized patient the use of Allen's method

of jejunostomy for feeding is ideal. In the subtotal gastrectomized patient both the decompression tube and the feeding tube are of tremendous importance to the safety and the comfort of the patient.

It seems that in some cases, at least, the creation of a gastric pouch is of definite benefit to the total gastrectomized patient. The pouch may be created by using the afferent and efferent loops of jejunum with very little difficulty, and may be done as a second stage procedure or at the original resection.

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PROLONGED SPINAL ANESTHESIA

Description of a Simplified Technique for Nupercaine

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SINCE the introduction of subarachnoid block by Bier in 1898, there has been a ceaseless search for a technique which would provide prolonged as well as controllable anesthesia. Prolonged spinal anesthesia may be obtained in one of two ways: (1) by the continuous or serial technique; (2) by a single intrathecal injection of a long-acting local anesthetic drug. The most recent advancement in the direction of prolonged anesthesia has been the introduction of the "continuous spinal" technique by Lemmon.[1] Although the continuous technique has been widely publicized and acclaimed, it is far from ideal. The thick cumbersome mattress, the frequent dislodgment of the indwelling needle, the difficulty and uncertainty of establishing anesthesia in the desired segment, and the possibility of injury to the intraspinal structures by either the needle or repeated doses of the drug are objectionable features. Besides, its induction is time consuming. The modified technique suggested by Tuohy,[2] employing the indwelling catheter, eliminates the mattress and failures due to dislodgment of the needle, but increases possibility of fistulas, infection, and leakage of spinal fluid.

Although many drugs have been employed for spinal anesthesia, few yield prolonged action by the single injection method. Pontocaine and nupercaine are the two currently available agents most useful in this respect. Pontocaine has enjoyed popularity since Sise[3] advocated its administration with glucose. The addition of glucose to solutions of local anesthetic drugs causes them to be hyperbaric or heavier than spinal fluid. The effects of gravity may then be utilized to extend anesthesia to the desired spinal segment by tilting the patient during the induction period. Exact localization of the drug is possible because cephalad diffusion of the pontocaine in the spinal canal appears to be minimized by the glucose. When employed by capable individuals in suitable subjects, the pontocaine-glucose technique yields excellent results and is entirely satisfactory. Although pontocaine is satisfactory for most operations, nupercaine is preferred when the procedure is to last more than two hours.

The various methods advocated for the administration of nupercaine are not as simple as the technique described by Sise for pontocaine. The method described by Jones[4] is the most popular at the present time. In this technique, a relatively large volume of a hypobaric solu-

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tion is introduced into the subarachnoid space. During the induction period, it is necessary to turn the subject face downward and tilt the table so that the head remains dependent for approximately ten minutes. The prone position insures a caudad, rather than cephalad, movement of the solution. It also allows the posterior root filaments to come into contact with the solution so that satisfactory sensory anesthesia is secured. The results obtained by Jones' technique are frequently disappointing because diffusion of the nupercaine is an uncontrollable factor. Satisfactory localization of the drug at the desired segment is secured with difficulty because there is no logical basis for determining the volume of solution required. Severe circulatory disturbances often follow the shift from the prone to the supine position after anesthesia is established. Besides, the induction is time consuming. The technique described by Wilson[5] also requires the use of a comparatively large volume of dilute solution. The subject remains in the upright posture during the injection so that upward diffusion is assured. This technique is characterized by lack of controllability and is fraught with considerable danger.

There is no reason why nupercaine cannot be substituted for pontocaine in the glucose technique. Pontocaine and nupercaine are used in approximately the same quantities as far as weight is concerned. Besides, both drugs behave similarly as far as solubility, stability, and pH of aqueous solution are concerned. The prolonged action of nupercaine could thus be secured with the precision and simplicity allowed by the pontocaine-glucose technique.

The use of hyperbaric solutions of nupercaine has been described in previous reports. Silverton[6] employed a solution of ½ per cent nupercaine in 6 per cent glucose. He relied principally upon barbotage to force the drug to the desired segment. Sankey and Whitacre[7] add glucose to the solution described by Jones. The height of anesthesia is controlled largely by the displacement afforded by the large volume of fluid, rather than by the effects of gravity.

The technique we have developed is simpler than any previously described for nupercaine. Furthermore, it is rapidly executed and is reasonably controllable. The results are little influenced by wide variations in such intrinsic factors as length and diameter of the cord, size of subarachnoid space, or body weight. There are six extrinsic factors which may affect the extent and intensity of spinal anesthesia. These are volume of solution injected, specific gravity of solution, rate of injection, dose of drug, site of injection, and position of the patient after injection of drug. The fewer the variations, the simpler the technique. In this technique all factors are fixed with the exception of position of the patient. The patient is tilted immediately after the injection and progress of anesthesia is followed from moment to moment until it is at the desired segment, at which time cephalad ascent may be terminated by leveling the table. If one desires, the drug may

be confined to the lowest spinal segments and anesthesia so localized that it involves the perineum only and not the legs. On the other hand, anesthesia may be established at the cervical segments if one so desires.

Nupercaine (also known as percaine) differs in a number of respects from procaine, pontocaine, and related anesthetic drugs. The majority of local anesthetic drugs are esters of complex alcohols. Nupercaine is a substituted amide derived from quinoline and is not an ester. The impression that nupercaine is derived from quinine is not correct. Quinine is a cupreine. Cupreines are complex structures which contain not only a quinoline nucleous but also other groups and radicals as well. Nupercaine is a weak base which forms salts when neutralized by mineral acids. The base is easily precipitated from aqueous solutions of its salts by alkalies and basic salts.

TECHNIQUE

The preparation employed for this technique consists of ½ per cent nupercaine hydrochloride dissolved in a phosfate buffer and saline solution. Each cubic centimeter contains 5 mg. of the drug. The volume of solution containing the estimated dose of nupercaine is thoroughly mixed with an equal volume of 10 per cent glucose in physiologic saline or distilled water. The resulting volume of solution, even when the maximum dose is employed, is relatively small. Variations in volume due to variations in dosage used are relatively insignificant. The mixture is injected into the subarachnoid space through a 20 or 22 gauge needle as rapidly as gentle pressure on the plunger permits without barbotage. Although the lateral prone position is preferred in performing the block, the upright sitting posture may be employed if one so desires. The spinal puncture is performed at the level of the third lumbar interspace for anesthesia above the costal margin and at the level of the fourth lumbar interspace when less extensive anesthesia is required. Immediately following the injection, the patient is placed on his back and the operating table is tilted in a Trendelenburg position. The angle varies with the height of the anesthesia desired. The head is flexed as sharply as possible upon the thorax by supporting it upon a pillow which has been doubled upon itself (Fig. 1). Unless this precaution is rigidly observed, the mixture may ascend into the cervical region and cause respiratory failure by affecting the phrenic nerves. Generally, within fifteen to twenty seconds the subject notes a feeling of numbness in the lower extremities. In contrast to the behavior of the shorter-acting drugs such as procaine, metycaine, monocaine, and others which seem to act almost immediately, the onset of anesthesia is delayed when nupercaine or pontocaine is employed. Occasionally, ten minutes or more may be required for establishment of complete anesthesia by nupercaine introduced intrathecally. However, hypalgesia, which always precedes anesthesia, ensues immedi-

ately. Consequently, when performing the sensory examination to establish the level of anesthesia, one does not seek the areas of complete insensibility, but instead those of hypalgesia or diminished sensation. An area of complete anesthesia will be revealed over the anterior surface of the thighs within approximately one minute after completion of the injection. One should always test sensation gently with a sharp instrument. As the examining instrument is advanced cephalad, an area of hypalgesia is encountered which appears to diminish in intensity and merges into a totally unanesthetized area over the upper part of the body. The line of demarcation between the hypalgesic and unanesthetized area is reasonably sharp and well defined. When the line of demarcation lies in the dermatome in which anesthesia is desired, the ascent of the drug is terminated by promptly

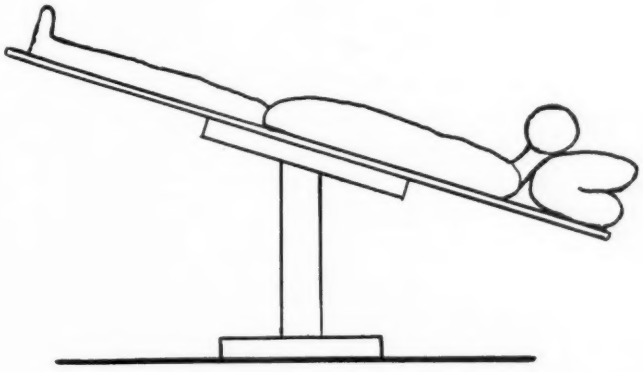

Fig. 1.—Method of acutely flexing the head to prevent movement of the drug into the cervical portion of the spinal canal.

readjusting the operating table to the horizontal position. The hypalgesic areas in due time become completely anesthetic. There appears to be little or no tendency for anesthesia to creep beyond a given level once it is established at that level. Under no circumstances does one rely upon the line of demarcation between the zone of complete anesthesia and the zone of hypalgesia for establishing the level. If one relies upon anesthesia rather than hypalgesia in the sensory examination, anesthesia may extend far above the desired spinal segment.

The time the patient remains in the inclined position varies for each individual case. As few as thirty seconds or as many as three minutes may be required for the drug to reach the desired segments. So many variable factors modify the cephalad progress of the drug that no fixed time interval can be stated. The degree of inclination of the operating table likewise cannot be standardized, but must be adjusted to suit the needs of each individual patient. For upper abdominal surgery the lower seven thoracic segments must be anesthetized. To secure this extent of anesthesia, the inclination must be steep—approximately twenty degrees. One should be prepared to brace the shoulders imme-

diately after the solution is injected. If the operating table is inclined at an angle of less than twenty degrees, the drug may not reach the thoracic spinal roots and unsatisfactory anesthesia results. For the sake of safety, the progress of the drug in the cephalad direction should be followed by almost continuous sensory examinations as long as the patient is in the inclined position. The operating table should be restored to the horizontal position immediately if the drug appears to advance rapidly or if anesthesia advances beyond the desired sensory segments. If the drug appears to involve the upper thoracic or lower cervical segments, the operating table should be restored to the horizontal or placed in the reversed Trendelenburg position at an angle of 5 to 10 degrees. The reversed Trendelenburg position not only terminates the upward advance of the drug, but favors a caudad regression of the solution. For best results, it is imperative that there be as little delay as possible in inclining the table when the intrathecal injection is completed. Failure to obtain satisfactory anesthesia for upper abdominal surgery is frequently due to delays in inclining the table promptly. The operating table need not be as steeply inclined when anesthesia below the costal margin is sought. An angle of 10 to 15 degrees is usually sufficient. To obtain "low" spinal anesthesia, that is, anesthesia of the perineum or extremities, the operating table should be tilted immediately to the Trendelenburg position at an angle of approximately 10 degrees for five to ten seconds. It is then returned to the horizontal position if anesthesia over the hypogastric area is desired and placed in the dependent position at an angle of 5 degrees if anesthesia is to be confined to the extremities. Failure to tilt the table to the Trendelenburg position immediately after the injection is completed may result in uneven or "spotty" anesthesia and incomplete muscular relaxation. It is obvious that this technique cannot be successfully employed if a table which is easily tilted is not available.

It is a well-established pharmacologic fact that the sensory fibers of a mixed nerve are the first to be affected by a local anesthetic drug, the autonomic fibers next, and the motor fibers last. For several minutes after establishment of sensory anesthesia by this technique, motor power remains unchanged or is mildly depressed. Motor paralysis is rarely completely established in less than five minutes. The degree of motor paralysis varies in the different spinal segments, depending upon the concentration of drug in each segment. In the thoracic region where the concentration of drug is obviously less than at the site of puncture, more sensory fibers are involved than motor. Muscle relaxation, therefore, is incomplete or absent. At the site of injection and throughout the lumbar region, the concentration of drug is greater and all types of nerve components are affected. Anesthesia, therefore, is complete. The concentration of drug in the upper spinal segments may be increased by increasing the total amount of drug injected.

Motor anesthesia becomes more extensive. If is obvious, then, that muscle relaxation is controlled by varying the quantity of drug. The dosage, therefore, depends upon intensity and extent of anesthesia desired. For upper abdominal surgery, 12 to 15 mg. of nupercaine yield satisfactory sensory and motor anesthesia. For anesthesia below the costal margin, 7 to 10 mg. are ample; for surgery of the extremities, 2½ to 5 mg. are sufficient. In the upper age groups, the lower limits of dose ranges should be employed. Formulas such as milligrams per pound of body weight, milligrams per centimeters of length of the vertebral column and the like have proved to be no guide to the amount of drug required. The intrinsic factors, such as length and diameter of the cord, and volume of the spinal subarachnoid space, are so variable that in using a drug which is potent and which is used in relatively small quantities, these rules of dosages are valueless.

The time-honored custom of mixing the drug with cerebrospinal fluid was dispensed with in this technique for two reasons: (1) Spinal fluid, which has a pH of 7.4, is sufficiently alkaline to cause a precipitation of nupercaine base from aqueous solutions of the hydrochloride. Unsatisfactory anesthesia results if one employs a solution in which flocculation has appeared. Laboratory studies suggest that glucose prevents precipitation of the free base when solutions of salts of local anesthetic drugs are injected into the subarachnoid space.[8] (2) The solution may be prepared in advance of the lumbar puncture and injected immediately upon completion of the puncture. Failures due to dislodgment of the needle or shifting of the patient's position while solutions are being prepared are thus averted.

Ten to fifteen minutes should be allowed to elapse after injection of the solution before changes in posture are attempted. Trendelenburg, prone, lithotomy, and other positions necessary for completing the operation may then be employed with safety. After ten minutes have elapsed, the glucose diffuses throughout the spinal fluid and no longer causes the drug to shift about in the subarachnoid space.

RESULTS

Anesthesia induced by this technique was employed for 800 consecutive surgical procedures of all types. In 9.5 per cent of the cases, anesthesia extended to or beyond the fifth thoracic segment; in 59 per cent of the cases, it was established between the twelfth and ninth thoracic segment; and in the remainder of the cases, it was confined exclusively to the lumbar and sacral segments. Satisfactory anesthesia was obtained in all but ten subjects. In these, failure was due to unsatisfactory lumbar puncture. In thirteen subjects undergoing abdominal surgery, sensory anesthesia was satisfactory, but motor anesthesia of the lower extremities was incomplete. The abdominal muscles, however, were well relaxed and there was no interference with the

progress of the operation. It is felt that this incomplete motor paralysis was due to a delay in tilting the operating table following the induction of the solution.

Duration of anesthesia ranged from two to three hours in 0.8 per cent of the cases, three to four in 98 per cent, four to six in 1.2 per cent of the cases. The average duration of anesthesia was three and one-half hours.

Although it is difficult to make comparisons, the general impression gained was that hypotension was no more severe or more prevalent with nupercaine than in spinal anesthesia with other drugs. It appeared, as one would expect, more frequently in "high" spinal than in "low" spinal anesthesias. In most instances, it appeared immediately following or shortly after induction of anesthesia. Vasopressor drugs such as ephedrine, neosynephrin, or enethyl were effective restoratives in every instance in which it occurred. Nausea, vomiting, and retching were observed in approximately 1 per cent of the cases. In each instance these reactions were observed during upper abdominal explorations, and appeared to be due to traction upon the viscera. Restlessness, which appears in the majority of subjects undergoing prolonged operations under spinal anesthesia, was controlled by the use of sedatives. A combination of morphine sulfate gr. $\frac{1}{6}$ to $\frac{1}{8}$, and scopolamine gr. $\frac{1}{150}$ to $\frac{1}{200}$ (a ratio of 25 parts morphine to 1 part scopolamine), intravenously, was found to be particularly useful for this purpose.

The technique was employed for anesthesia for abdominoperineal resection in seven subjects. This is noteworthy because the hypobaric solution of nupercaine has not proved satisfactory for this type of operation. The abdominal anesthesia is satisfactory, but the sacral and lower lumbar segments frequently are not completely anesthetized. This discrepancy has not been adequately explained. To insure satisfactory anesthesia in the perineal area, the injection may be made while the patient is in the upright sitting position. The patient is then tilted and managed as previously described.

DISCUSSION

In determining the value of a local anesthetic drug for spinal anesthesia, one must base its merits upon potency, systemic toxicity, and local toxicity. Objections are frequently raised to nupercaine for spinal anesthesia on the grounds that it is more toxic than many of the currently employed local anesthetic drugs. Nupercaine is both more toxic and more potent than procaine and similar agents. Too often toxicity is confused with potency. The comparative systemic toxicity of local anesthetic drugs for man is difficult to estimate. Studies in animals indicate that nupercaine is approximately eight to ten times more toxic than procaine.[9] On the other hand, it is twelve to fifteen times more potent than procaine. One must bear in mind

that potency and toxicity do not necessarily parallel each other. One milligram is theoretically equivalent in action to approximately 15 mg. of procaine. The amount of nupercaine required for the same degree of anesthesia yielded by procaine is considerably less. Therefore, though nupercaine possesses a greater absolute toxicity, its relative toxicity approximates that of procaine.

The more potent one drug is, compared to another, the greater the intensity of anesthesia it will cause when used in equivalent amounts. Therefore, the intensity of anesthesia may be controlled by varying the dose, regardless of the agent employed. The severity of the physiologic disturbances which accompany spinal anesthesia depends upon the intensity of anesthesia. The greater the number of dermatomes affected by the drug and the greater the number of sensory, motor, and autonomic components which are involved in a spinal segment, the more intense will be the anesthesia. Physiologic disturbances appear during spinal block, regardless of the drug employed, if the amount injected is sufficient to produce extensive anesthesia. The most distressing physiologic disturbance accompanying spinal anesthesia is hypotension. Although there is no agreement regarding the mechanism causing it, it is probably the result of a combination of factors, all of which are dependent upon the extent of the resulting paralysis. The relaxation of the muscles of the extremities, the paralysis of the abdominal muscles, the diminution of ventilation from paresis of the intercostal muscles, and the paralysis of the sympathetic nervous system, all contribute to the peripheral circulatory failure and cause pooling of the blood in the venules and capillaries. Relatively small amounts of nupercaine, by yielding extensive anesthesia, may cause marked physiologic disturbances, particularly when the anesthesia affects many spinal segments. Hypotension is a manifestation of potency rather than toxicity. A toxic reaction is due to a high concentration of drug in the blood. Such reactions may be due to accidental intravascular injections or rapid absorption of the drug into the blood stream. Excitement, nausea, vomiting, convulsions, and ultimately respiratory and circulatory failure are the usual symptoms of toxicity. Local anesthetic drugs cause, at first, a marked stimulation of the central nervous system. If the overdosage is massive, depression and paralysis quickly follow. Toxic reactions are not benefited by vasopressor substances, oxygen, and other supportive measures so useful for the relief of hypotension. Fortunately, toxic reactions are rare in spinal anesthesia.

Considerable disagreement exists concerning the reversibility or "local toxicity" of various local anesthetic drugs administered intrathecally. Neurologic complications ascribable to changes produced in the intraspinal elements by the local action of the drug have been reported following the use of most of the currently employed local anesthetic drugs. It is almost impossible to draw any conclusion regarding the

relative merits of one agent over another from the isolated case reports which appear in the literature. Systematic studies of this problem have not been attempted and are few because of the difficulty in studying the spinal cord in routine autopsies. A considerable amount of data of an experimental nature exists, however. Recently Co Tui and his co-workers,[10] in comparing the effects of procaine, nupercaine, and monocaine in cats and rabbits, found that slight transitory changes occur in the cords of these animals when therapeutically equivalent doses of drugs are employed. The quantity of nupercaine required to produce the same anesthetic effect as the pharmacologic equivalent of procaine produced the same degree of histologic change as procaine. The changes lasted for the same period of time with each drug. However, when the minimum lethal dose was employed, the changes produced by nupercaine were more intense and not reversible. On this basis, one would assume that the local toxicity of nupercaine is no greater than a therapeutic equivalent of procaine. No instance of a neurologic complication was observed in any case in this series during the period of hospitalization.

Obviously, the same criteria for selection of patients, contraindications, conduct, and precautions employed for spinal anesthesia with other drugs are applicable to this technique.

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PULMONARY COMPLICATIONS FOLLOWING APPENDECTOMY AND HERNIORRHAPHY

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THE pulmonary complications following 400 operations for hernia and 361 operations for appendicitis in the Station Hospital, Camp Rucker, Ala., are shown in this paper. An insignificant number of the cases of appendicitis were in women and all of the hernias were in men. This is a report of pulmonary complications following lower abdominal operations on healthy young men between 18 and 40 years of age, using spinal anesthesia (150 mg. novocain) as the routine anesthetic, from July, 1942, until May 15, 1944.

The differentiation of atelectasis, bronchitis, pneumonitis, and bronchopneumonia is dependent upon clinical observation with roentgenographic studies. Spink and Bellis[1] have noted that it is not always possible to be sure of the exact pathology in the early and minimal cases. King[2] believes that the presence of a bronchitis is the cause of the secretion that accumulates in the bronchial tree and may result in atelectasis. King has shown that the incidence of pulmonary complications varies with the interest taken in the subject, reporting 3.7, 5.2, and 6.8 per cent being present during successive years. Haight and Ransom[3] state that atelectasis is most common after upper abdominal surgery, that it is twice as frequent in men as in women, that it is twice as frequent after gastric surgery as it is following biliary surgery, and that it occurs more frequently in individuals with a chronic cough, chronic bronchitis, sinusitis, asthma, or an acute cold. Oswald[4] found the incidence twice as high in those over 40 years of age, an incidence of 5 per cent after hernia and appendix operations, and an incidence of 20 to 30 per cent after upper abdominal surgery. Maier[5] reports an incidence of 10 per cent of atelectasis in the pulmonary remnant after lobectomy for bronchiectasis without a fatality, but states that Gowar reported 33 per cent atelectasis in 154 cases of lobectomy for bronchiectasis, and that one-third of the deaths were due to suppuration in the unexpanded portion of the lung.

Table I shows that there were 35 patients (4.6 per cent) with pulmonary complications, following 761 operations for hernial repair or some type of appendicitis.

There was an incidence of 5.5 per cent (or 22) pulmonary complications after 460 herniorrhaphies. Thirteen (or 3.6 per cent) pulmonary

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TABLE I

TYPE OF OPERATION	NUMBER OF OPERATIONS	NUMBER OF PULMONARY COMPLICATIONS	PER CENT
Total operations for hernia or appendicitis	761	35	4.6
Total hernia operations	400	22	5.5
Bilateral inguinal hernia	101	12	11.9
Right inguinal hernia	166	3	1.8
Left inguinal hernia	95	7	7.4
Total operations for appendix pathology	361	13	3.6
Operations for gangrenous appendix	28		
Perforated appendix	14		
Appendical abscess	7		
	49	5	10.4
Operations for simple appendicitis	312	8	2.6

complications followed 361 operations for appendicitis. There was an incidence of 11.9 per cent following 101 bilateral hernias, an incidence of 1.8 per cent following 166 right inguinal herniorrhaphies, and an incidence of 7.4 per cent following 95 left inguinal herniorrhaphies. The incidence was 10.4 per cent after 49 operations for gangrenous or perforated appendicitis or appendical abscess.

Table II shows that atelectasis alone was present in 12 cases and atelectasis with pneumonitis was present in 5 cases. Bronchopneumonia was present 4 times, bronchitis 13 times, and a lobular infarct occurred in the left base in 1 case. Lobar atelectasis was present in 10 cases, one-half of these being in the left lower lobe. Massive atelectasis occurred once in the right lung and once in the left lung.

TABLE II

TYPES OF COMPLICATIONS

Atelectasis alone			12 (Right lung, 5; left lung, 7)
Upper lobe		3	
Right	2		
Left	1		
Lower lobe		7	
Right	2		
Left	5		
Massive		2	
Right	1		
Left	1		
Atelectasis with pneumonitis		5	
Bronchopneumonia		4	
Bronchitis		13	
Lobular infarct left base		1	

Table III shows that atelectasis was present in 12 of the 22 patients with complications following hernia operations and was present 5 times in 13 patients with complications following operations for appendicitis.

Spinal anesthesia was used routinely and there were 32 (or 4.8 per cent) complications after 671 spinal anesthetics. There were two (or 3.9 per cent) complications following 51 spinal anesthesias supplemented with general anesthesia. It is generally felt that the anesthetic is not a

TABLE III

Complications following hernia operations	
Atelectasis alone or with pneumonitis	12
Bronchopneumonia or bronchitis	10
Total	22
Complications following appendicitis operations	
Atelectasis alone or with pneumonitis	5
Bronchopneumonia or bronchitis	7
Lobular infarct	1
Total	13

great factor in causing pulmonary complications or that spinal anesthesia will prevent pulmonary complications.

There was no mortality in the 761 cases. Delay in aeration of an atelectatic lobe occurred in two cases. In one case the first film was made twenty-four hours postoperatively (Aug. 27, 1943) and showed a beginning atelectasis. Under routine treatment including sulfadiazine there were no symptoms or fever that could not be explained by the abdominal condition. However, an x-ray view taken Sept. 6, ten days postoperatively, shows a well-developed right, upper lobar atelectasis. This was not producing any symptoms or fever. An x-ray taken September 13 showed a marked clearing of the process and the lung was entirely clear September 27. There were no patients with lobar pneumonia. A small infarct occurred in one case in the left base, obliterating the costophrenic angle and causing a density on the radiograph that was reported present one month after discharge from the hospital, but not producing any symptoms.

Prevention of pulmonary complications is possible by attention to many details that influence and predispose to its occurrence. In one-third of our cases there was a history of respiratory infection, tooth infection, or sinusitis immediately prior to, or at the time of, operation. The eradication of infection in the nose, mouth, or respiratory tract and lapse of period of 7 to 14 days before operation is desirable. An acute phase of a chronic pulmonary infection in the presence of bronchiectasis should be allowed to subside completely. The pulmonary bronchial system before, during, and after the operation should be kept clear. For these lower abdominal cases the patient was asked to clear his bronchi prior to operation and a slight Trendelenburg position was used during the operation. The patients were usually ventilated at the end of operation with carbon dioxide and the importance of deep breathing, coughing, expectorating, and moving the legs was stressed. They were encouraged to cough, and were assisted in coughing. When bronchial secretion was obviously present and the patient was having difficulty expectorating, carbon dioxide oxygen ventilation was given twice daily. Sulfadiazine, 1 Gm., with sodium bicarbonate, 1 dr., was given six times daily with a fluid intake sufficient to give a urinary output of 1,000 c.c. or more.

Very little has been written about the psychologic inhibition of coughing that has played a part in the development of atelectasis in many of our cases, especially in the cases of massive atelectasis encountered at the onset of the series. Coughing causes a pain in the wound and the soldier thinks of tearing the repair with the development of another hernia, which is a real, tangible, experience, whereas he probably has never heard of pulmonary complications. In the great majority of cases this is easily overcome by explanation before operation, repeated during the operation by the anesthetist, and by frequent visits, reassurance, and personal contact postoperatively. Not the least of this reassurance is aiding the patient in coughing by holding his wound while he coughs and at times giving a few sharp slaps on the back. To reinforce this psychotherapy and actually stimulate deep breathing, carbon dioxide and oxygen inhalations have been used frequently for a few days. This is of more value after herniorrhaphy since two weeks flat in bed, or more, are necessary postoperatively, whereas with the simple appendectomies with a McBurney incision the patient is given freedom to do as much as he will and encouraged to early sitting and walking.

The treatment of atelectasis and bronchitis is usually satisfactory with the use of inhalations, sulfadiazine, and more energetic working with the patient for him to cough up the secretions. Frequent radiographic studies, at least daily for a few days, are necessary to establish the aeration status of the lungs. If conservative measures are not successful, aspiration of the plugs and establishment of a clear airway are necessary. Haight and Ransom recommend the use of a soft No. 16 French urethral catheter when frequent aspirations are needed, and the patient is not cyanotic. Bronchoscopic aspiration is necessary if these measures are not successful.

SUMMARY

There were 35 (or 4.6 per cent) pulmonary complications following 761 operations for hernias and for pathologic conditions resulting from disease of the appendix in a station hospital during the past two years. There was no mortality following these operations.

Pulmonary complications were present in 10.4 per cent of 49 operations for severe appendicitis and 2.6 per cent of 312 operations for uncomplicated appendicitis.

There were 22 (or 5.5 per cent) pulmonary complications after 400 operations for hernia.

Atelectasis was present alone or in combination in 17 cases and bronchitis or bronchopneumonia was present in 17 cases. There was one small pulmonary infarct.

We do not believe that the use of spinal anesthesia is a factor in the development of postoperative pulmonary complications.

The presence of infection of the upper respiratory tract, sinuses, teeth, tonsils, or the lungs will increase the development of pulmonary complications.

Prevention of atelectasis is best accomplished by the preservation and activation of the normal cough by reassurance, adequate explanation of the objective to the patient, and the employment of turning, deep breathing, slapping on the back, and CO_2 inhalations as adjuvants of psychotherapy all combining to result in normal expectoration by the patient of the bronchial secretions.

Sulfadiazine and sodium bicarbonate are used routinely with pulmonary complications because of the frequent impossibility of differentiating infection from atelectasis early in the process and because atlectasis is frequently associated with infection.

Catheter aspiration or bronchoscopy is advocated if conservative measures fail.

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CARDIOVASCULAR DISTURBANCES FOLLOWING PNEUMONECTOMY

An Attempt to Correlate Blood Pressure and Intrapleural Pressure Changes

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CARDIOVASCULAR disturbances during and immediately after pneumonectomy have been noted by many observers and have been the object of considerable attention. The problem is no less complex than the whole subject of cardiovascular physiology. Many factors have been suggested as being responsible for these changes; many factors, undoubtedly, operate.

In our experience a considerable decrease of blood pressure is commonly noted immediately after removal of a patient from the operating table to the bed in his room. Maier[1] suggested that after pneumonectomy, changes in intrapleural pressure incident to change in the position of the body of the patient may be responsible for sudden drops in blood pressure and changes in the character of the pulse. In order to test this hypothesis and in an attempt to throw more light on this problem, we have measured, in five cases, intrapleural pressure in three positions immediately after closure of the thoracic wall following pneumonectomy, and have taken simultaneous records of the blood pressure and the pulse rate.

Frequency, Magnitude, and Duration of the Fall of the Blood Pressure of Patients Immediately After Pneumonectomy

The records of twenty-eight patients on whom pneumonectomy had been performed were studied to determine the frequency and magnitude of a significant fall of blood pressure on the return of the patient from the operating room to his hospital room. Of these twenty-eight, seventeen (61 per cent) had a significant drop (average 35 mm. of mercury).

The duration of the abnormal reduction of blood pressure varied widely, with forty minutes the minimum and forty-eight hours the maximum. The average time required for return of the pressure to approximately normal (that is, preoperative) levels was 9.6 hours.

In order to establish some basis for comparison, nine cases in which transthoracic exploration for inoperable carcinoma of the cardia of the stomach or of the lower end of the esophagus had been performed and nine cases in which partial gastrectomy for carcinoma or for duodenal ulcer had been performed were used to determine the frequency and magnitude of decreases of blood pressure in the same immediate postoperative period.

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In the group of cases in which transthoracic exploration was performed, six patients had no appreciable change in blood pressure during this period. Three had transitory falls to 70 to 90 mm. of mercury systolic, but their blood pressure rose to normal in the next one to three hours.

In the second group of nine cases in which partial gastrectomy was performed, only one patient had an appreciable fall in pressure. This was a man aged 64 years, whose pressure fell from 144/80 to 110/64, but steadily and rapidly rose to its preoperative level. There was little in his history to suggest heart disease but he died of coronary occlusion on the fourth postoperative day.

To summarize, in only four of eighteen cases in which rather extensive surgical procedures had involved the thoracic and abdominal cavities was there an appreciable fall of blood pressure between the departure of the patient from the operating room and his arrival in the hospital room. In no case was the fall great nor was it of long duration. This result is in rather striking contrast to the group in which pneumonectomy was performed.

DESCRIPTION OF METHOD

Intrapleural pressures were measured by means of a U manometer (water) immediately on closure of the thoracic wound in five cases in which pneumonectomy had been performed.

In four instances pressures were measured through a 24 F. rubber catheter, which had been allowed to remain in the wound during closure. Gentle suction through the catheter was applied throughout the procedure of closure in order to prevent the development of tension pneumothorax by the valve action of the intercostal structures from the time they were loosely brought together preparatory to suture until the thoracic wall was closed in an airtight manner by suture. In each case the thorax was closed securely before pressure readings were taken and the first intrapleural pressure was nearly the same as atmospheric pressure.*

With the patient successively in the lateral horizontal position, in the dorsal horizontal position, and in the dorsal position with the head of the table elevated 30 degrees, intrapleural pressures were measured. The patient had remained in the lateral position with the pneumonectomized side up throughout the course of the operation and the first measurement was made before this position was altered. Simultaneously, the blood pressure and pulse rate were determined.

Intervals between measurements in the three positions varied from one to three minutes, or the time, in each instance, consumed in changing the patient's position and in making the necessary measurements.

*There was one exception, in Case 2, in which because of failure to regulate suction considerable negative pressure developed.

PREOPERATIVE MEDICATION

In each case preoperative medication had been the same: 1½ gr. (0.1 Gm.) of soluble pentobarbital (nembutal) on the evening before operation, 1½ gr. (0.1 Gm.) of soluble pentobarbital at 7 A.M. on the day of operation, and ⅙ gr. (0.01 Gm.) of morphine sulfate and 1/150 gr. (0.00043 Gm.) of atropine sulfate approximately one hour before the induction of anesthesia.

ANESTHETIC AGENTS EMPLOYED

In every case anesthesia was induced with nitrous oxide and oxygen, to which mixture ether was added subsequently. The mixture was administered with a closed system through an intratracheal tube.

INTRAPLEURAL PRESSURE RECORDS

Depending on the surgical findings regarding the state of the pleura and the consequent mobility of the mediastinum, the cases could be divided into two groups: (1) those with thickened pleura and an immobile (fixed) mediastinum (two cases); and (2) those with thin pleura and a mobile mediastinum (three cases).

In those of the first group, there were no significant changes (that is, changes that could be measured accurately with this method) in intrapleural pressure with changes in position.

In the second group, in which the mediastinal pleura had appeared grossly normal and in which the mediastinal mobility had been apparent with respiratory motion while the thorax was open, there was a considerable rise in intrapleural pressure with the change from a lateral horizontal to a dorsal horizontal position and a lesser but measurable change to a lower pressure with elevation of the head of the table 30 degrees.

The position of the mediastinum as shown by the roentgenogram of the thorax made immediately after operation was considered as reflecting the pressure differential between the two sides of the thorax (if the mediastinum was not rendered immobile by pleural thickening). In the absence of atelectasis in the remaining lung, shift of the mediastinum away from the side of the pneumothorax invariably meant an abnormally high intrapleural pressure on that side. Thus, in a number of cases in which measurements of intrapleural pressure had not been made, abnormal position of the mediastinum in an early postoperative roentgenogram of the thorax gave indirect evidence of significant deviation of intrapleural pressure from the normal and so afforded observations pertinent to this study.

CORRELATION OF INTRAPLEURAL PRESSURE, BLOOD PRESSURE, AND PULSE RATE

In those cases (Cases 1 and 2) in which the pleura was thickened and the mediastinum was thus rendered immobile, our measurements failed to show any significant change in intrapleural pressure with changes in

position. Yet in each instance there were minor drops in blood pressure during these maneuvers and a considerable fall in blood pressure between the time when it was last recorded immediately before departure from the operating room and the time when it was first recorded on arrival in the patient's room ten to twenty minutes later. In both cases there was a slight increase in the pulse rate with the fall in blood pressure. Within a few hours after return to the hospital room, the blood pressure gradually rose to normal preoperative levels and the general condition of the patient was never considered to be alarming. Intrapleural pressure had been adjusted to approximately atmospheric levels and the mediastinum was shown by roentgenogram to be in the midline when the patient was lying on his back.

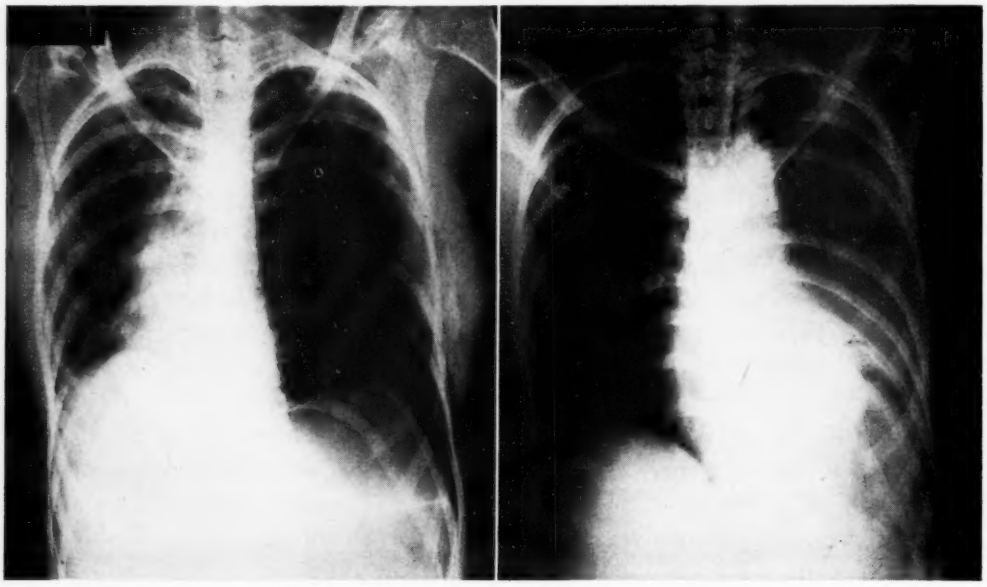
In Cases 3, 4, and 5, in which the pleura appeared normal and the mediastinum mobile, there were changes in the intrapleural pressure with changes in the position of the patient. These changes were of similar magnitude in each of the three cases. In Case 3 there was no measurable change in blood pressure or pulse rate during these maneuvers. In Case 4 there was a fall in systolic pressure of 5 mm. of mercury and in diastolic pressure of 10 mm. without significant change in the pulse rate. These changes of blood pressure disappeared within a few minutes, before the patient could be removed from the table. In Case 5 the reaction was similar to that in Case 4 except for a slight slowing of the pulse during the period of pressure measurements.

In each of these three cases there had been a considerable rise of intrapleural pressure in the empty pleural cavity with change from a lateral to a dorsal position, but in no case was there any considerable difference between the blood pressures or the pulse rates as recorded in the two positions. Postoperative roentgenograms of the thorax with the patient in the dorsal horizontal position showed the mediastinum in the midline.

Observations on the blood pressure and pulse rate during the same period were made in three other cases (Cases 6, 7, and 8) in which intrapleural pressure measurements were not made but in which immediate postoperative thoracic roentgenograms gave indirect evidence on intrapleural pressure. It has been suggested that changes of intrapleural pressure are important chiefly because of their effect on the position of the mediastinum. In Case 6 there was a drop from 105/70 to 85/60 when the patient was turned on her back and held in a head-and-shoulders-up position (propped up approximately 30 degrees) while a dressing was being applied. Thirteen minutes later the pressure had returned to 110/75 while she was lying on her back. Immediate postoperative roentgenograms in this position showed the mediastinum in the midline. Elevation of the head rather than the change from the lateral to the dorsal position seemed to be responsible for the transitory fall of blood pressure. In Case 8 there was no change of blood pressure during this maneuver, although there was

some fall subsequently during bronchoscopic aspiration. In this case a thoracic roentgenogram showed a slight shift of the mediastinum to the good side but of such minor degree that no adjustment of the intrapleural pressure was deemed necessary.

Case 7 is particularly interesting and pertinent to this study. Suction was not applied to the pleural space during closure and an abnormally high intrapleural pressure was inadvertently allowed to develop with a resulting considerable shift of the mediastinum away from the



A. *B.*

Fig. 1 (Case 7).—*A.* The thorax immediately after pneumonectomy. The considerable shift of the mediastinum to the right is apparent. *B.* A few hours later, after the withdrawal of air from the side of the pneumothorax and the adjustment of intrapleural pressure to approximately atmospheric level (see Fig. 2). Both views were made with the patient lying flat on her back.

side of the pneumothorax. This was discovered in the routine thoracic roentgenogram taken on arrival of the patient in her room. Two hundred cubic centimeters of air were withdrawn without visible change in the position of the mediastinum or change in the blood pressure or pulse rate. With the pneumothorax apparatus an additional large volume (approximately 700 c.c.) of air was withdrawn and the pressure adjusted to approximately atmospheric level. This required about five minutes. There was no change in the blood pressure or pulse rate, although the respiratory rate fell almost immediately from 24 per minute to 16 per minute. A roentgenogram of the thorax taken a few minutes later showed the mediastinum to have returned to its normal midline position (Figs. 1 and 2).

Of the eight patients just considered, two had mediastinal shift: one without a fall in blood pressure; the other with a slight fall in

blood pressure, which was unaffected by relief of the tension pneumothorax and the return of the mediastinum to the midline. Of the six patients without mediastinal shift, three had known changes in intrapleural pressure associated with change in position of the body but without demonstrable effect on the blood pressure or pulse rate. Though there was no apparent correlation of the blood pressure and pulse rates with changes in intrapleural pressure, a fall of blood pressure was demonstrated in six of the eight cases during the period of return of the patient from the operating room.

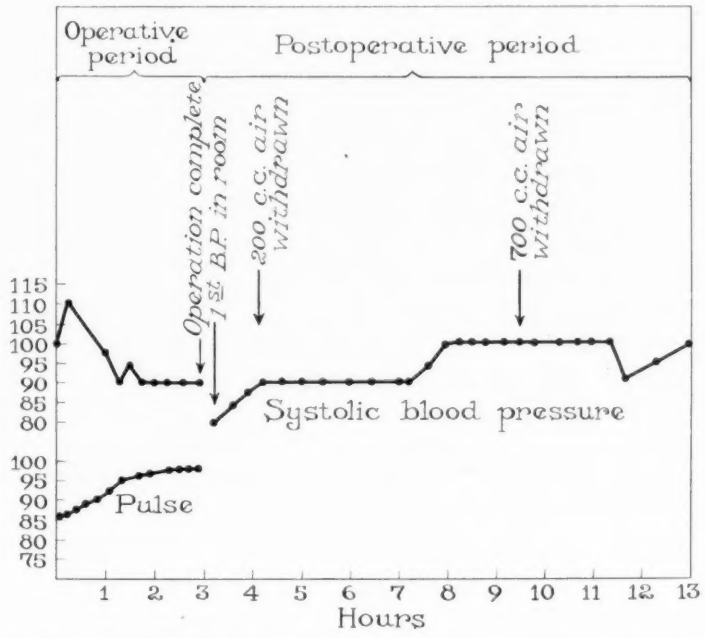


Fig. 2.—Systolic blood pressure during the operative and early postoperative period in Case 7. Time at which tension pneumothorax was relieved by the withdrawal of 700 c.c. of air is indicated. The failure of relief of pneumothorax to influence systolic blood pressure is apparent. Although the pulse rate for the postoperative period is not shown here, it too was unchanged.

It has already been pointed out that in a series of twenty-eight cases of pneumonectomy done prior to this study, there was a considerable postoperative fall of blood pressure in 61 per cent, so that the proportion in the combined series is 64 per cent. A further analysis of the series of twenty-eight cases on the basis of the presence or absence of mediastinal shift is of some interest. Roentgenograms of the thorax made immediately after operation showed the mediastinum in the midline in sixteen and with a slight shift (always to the good side) in twelve. In nine of the group of sixteen cases in which the mediastinum was in the midline, a fall of blood pressure occurred in the immediate postoperative period. The average fall was 32 mm. of mercury systolic and the period required for return to normal varied from two to thirty hours (Table I).

TABLE I

DATA ON TWENTY-EIGHT CASES IN WHICH PNEUMONECTOMY WAS PERFORMED

CHARACTERISTIC	POSITION OF MEDIASTINUM AS SHOWN BY IMMEDIATE POSTOPERATIVE ROENTGENOGRAM			
	MEDIASTIUM IN MIDLINE		MEDIASTINAL SHIFT	
	NO POSTOPERATIVE FALL OF BLOOD PRESSURE	POSTOPERATIVE FALL OF BLOOD PRESSURE*	NO POSTOPERATIVE FALL OF BLOOD PRESSURE	POSTOPERATIVE FALL OF BLOOD PRESSURE†
Total patients	7	9	4	8
Male	6	8	3	7
Female	1	1	1	1
Side of pneumonectomy				
Right	5	6	1	3
Left	2	3	3	5
Age of patient (years)				
10 to 19		1	1	
20 to 29	2			
30 to 39			1	1
40 to 49		6		1
50 to 59	3	1	2	4
60 to 69	2	1		2

*Average fall 32 mm. of mercury systolic. Blood pressure returned to normal levels in two to thirty hours.

†Average fall 44 mm. of mercury systolic. Blood pressure returned to normal levels in forty minutes to forty-eight hours.

In twelve cases there were minor degrees of shift of the mediastinum. In eight of this group there were immediate postoperative decreases of blood pressure averaging 44 mm. of mercury systolic and requiring forty minutes to forty-eight hours for return to normal preoperative range. In four cases in which there was mediastinal shift there was no significant change of blood pressure (Table I).

The side on which pneumonectomy was performed seemed to have no influence on this phenomenon; age was apparently not a factor of importance (Table I).

FACTORS INFLUENCING BLOOD PRESSURE AND PULSE RATE DURING AND IMMEDIATELY AFTER PNEUMONECTOMY

Loss of blood may be considered briefly as a possible factor in this phenomenon of fall of blood pressure. Considerable loss of blood is, in many of these cases, inevitable but an attempt was made in each case to compensate for loss of blood by transfusion of whole citrated blood during the operative procedure. The amount of blood given was determined by an estimation of the amount of blood lost and by the stability of the blood pressure and pulse rate during the operation. There was usually little change in the concentrations of hemoglobin estimated preoperatively and in the early (after forty-eight hours) postoperative period, indicating that replacement had about equaled blood lost. In no case of this entire group (thirty-six) was there postoperative evidence of gross hemorrhage into the empty pleural space. When it seemed advisable to remove fluid which had accumulated in the pleural space some days after pneumonectomy, this fluid was uni-

formly found to be grossly bloody but it was thin and lacked the appearance of whole blood. It was considered in each case to be a bloody effusion.

Observations on three patients suggested that lack of oxygen may have played a role in the temporary fall of blood pressure. The patient in Case 2 arrived in his room at 2:25 P.M., when his blood pressure was 85 mm. of mercury systolic and 52 diastolic. At 2:45 it was still 88 mm. of mercury systolic and 50 diastolic. In five more minutes he was placed in an oxygen tent and twenty-five minutes later the pressure had risen to 104 mm. of mercury systolic and 60 diastolic. In Case 6 the systolic pressure remained about 100 mm. of mercury during the first twenty-four hours. At the end of this time the patient was taken out of the oxygen tent for one hour, during which she became quite pale, her nail beds became slightly cyanotic, and her blood pressure promptly fell from 100/60 to 66/40. After her return to the oxygen tent, the pressure rapidly rose to the previous level. Change in her position in bed seemed to have no effect on her blood pressure. At the completion of the operation in Case 8 the blood pressure was 115 mm. of mercury systolic and 70 diastolic, the pulse rate 132 per minute, and the pulse was regular. Immediately after bronchoscopic aspiration, the pressure was found to have fallen to 95 mm. of mercury systolic and 60 diastolic, and the pulse had become more rapid and somewhat irregular. The patient's condition rapidly improved with the administration of oxygen by mask.

There is another consideration of some interest in this connection. During the final steps in the closure of the thoracic wall, the administration of the anesthetic agent is usually discontinued but the administration of oxygen by mask is continued until bronchoscopic aspiration is carried out. Immediately after this procedure, oxygen is again given for a few minutes until the patient's color has returned to normal. The period of his transportation from the operating room to the hospital room and until the first determination of blood pressure is made is usually of fifteen to twenty minutes' duration. During this period no oxygen was administered; otherwise the conditions under which the patient existed were no different from those during the final twenty to thirty minutes spent on the operating room table. Perhaps the return toward consciousness with recovery from the anesthetic lessens neural thresholds generally and permits the initiation of cardiovascular reflexes which are responsible for these changes of the blood pressure and pulse rate. These reflexes will be considered later. There are at least two considerations opposed to this argument: one is that the patient is still in a rather deep plane of anesthesia and usually does not begin to respond to external stimuli for another hour or more; the other is that in the experimental animal cardiovascular reflexes have been shown to exist during deep surgical anesthesia.

There are so many factors which directly or indirectly influence the blood pressure and the pulse rate that these observations suggesting a possible role for anoxia must be offered with caution. However, it is known that in man anoxia can cause a lowering of the blood pressure,[2] an increase in the rate and frequency, and an irregularity in the rhythm of the heart beat.[3] Maier and Cournand[4] found a reduction in the arterial oxygen saturation following pneumonectomy. This reduction lasted for several days. None of their determinations of arterial oxygen saturation were made, however, in the early postoperative period while the influence of the anesthetic agent remained. We have no direct data on the oxygen content of arterial blood during that period but our observations, together with the experimental work just cited, support the possibility that anoxia may be present in a significant degree at this period and that it may play a causative role in the lowering of the blood pressure.

The part played by cardiovascular reflexes initiated at the pulmonary stump and mediated through the vagus nerve during the recovery from anesthesia has previously been alluded to. Reflex disturbances initiated by operative manipulation and by direct electrical stimulation of the structures in the root of the lung have been experimentally investigated most recently by O'Shaughnessy.[5] He found that blocking the vagus was not a safeguard against such reflex disturbances. Respiratory reflexes were thereby abolished but cardiovascular reflexes remained. No immediate changes in pulse, blood pressure, or respiration have been noted on application of clamps to structures in the root of the lung in this present series of cases in which pneumonectomy has been performed. Arce and Brea[6] stated that vascular reflexes of this origin "may be manifested by sudden drop in blood pressure, bradycardia, extrasystoles, auriculoventricular dissociation, and ventricular fibrillation. . . . The intensity, degree, and severity of these phenomena vary considerably and cannot be foreseen."

As has been previously noted, these same cardiovascular effects can be produced by serious degrees of anoxia. The true role of either anoxia or vagal reflexes in the production of cardiovascular disturbances associated with pulmonary resection has not yet been established. Certainly, as suggested by O'Shaughnessy, unrecognized and unrecognizable reduction of cardiac reserve in some of these cases must exist preoperatively and be a factor in this phenomenon. It seems safe to say that slight to moderately abnormal intrapleural pressure in the side of the pneumothorax with mediastinal shift does not play an important part.

SUMMARY

Rather severe fall of blood pressure immediately following pneumonectomy has been noted commonly. In 64 per cent of a series of thirty-six cases herein reported such a drop occurred. Abrupt changes of the intrapleural pressure on the side of the pneumothorax, which were

produced by changes in the position of the body of the patient, were investigated as a possible cause.

Determinations of intrapleural pressure were made on five patients immediately after closure of the thorax after pneumonectomy. These determinations were made with the patient in three positions. Simultaneous records of the blood pressure and pulse rate were kept. Records of a number of other patients were reviewed for data pertinent to this study.

In cases in which the mediastinum was mobile, changes in the position of the patient produced changes in the intrapleural pressure, but no constant change in the blood pressure or pulse rate could be correlated with this change.

As factors in this postoperative lowering of the blood pressure, hemorrhage, anoxia, and cardiovascular reflexes mediated through the vagus were briefly considered.

REPORT OF CASES

CASE 1.—The patient was a man, aged 42 years. On April 6, 1944, left pneumonectomy was performed for saccular bronchiectasis secondary to multiple pulmonary abscesses. The pleural cavity was completely obliterated but adhesions were not dense. The pleura, including the mediastinal pleura, was definitely (⅛ inch [0.3 cm.]) thickened. The mediastinum appeared immobile.

Immediately after closure of the wound the intrapleural pressure was determined by means of a 15 gauge needle inserted in the fifth interspace in the midaxillary line. At 1 P.M., while the patient was lying on his right side, the blood pressure was 110 mm. of mercury systolic and 65 diastolic; the pulse rate was 100 per minute. The intrapleural pressure was –4 cm. of water on inspiration and +8 cm. on expiration. At 1:02, while the patient was lying on his back in a horizontal position, the blood pressure was 100 mm. of mercury systolic and 60 diastolic; the pulse rate was 100 per minute. The intrapleural pressure was –4 cm. of water on inspiration and +8 cm. on expiration. At 1:05, while the patient was lying on his back, with his head and shoulders elevated approximately 30 degrees, the blood pressure was 100 mm. of mercury systolic and 60 diastolic, and the pulse rate was 104 per minute. The intrapleural pressure was –2 cm. of water on inspiration and +6 cm. on expiration.

At 1:20 P.M. the first determination of blood pressure made after the patient's return to his room showed a systolic pressure of 80 mm. of mercury and a diastolic pressure of 40. The pulse rate was 110. In four hours the blood pressure rose to 110/60 and the pulse rate fell to 90 per minute. Both remained stable thereafter.

A roentgenogram of the thorax made two hours after operation showed no mediastinal shift.

One thousand cubic centimeters of whole citrated blood were given during the operation. The concentration of hemoglobin was 12.7 Gm. per 100 c.c. of blood before the operation and 13.7 Gm. forty-eight hours after the operation.

CASE 2.—The patient was a man, aged 50 years. On April 13, 1944, left pneumonectomy was performed for bronchogenic carcinoma. There were dense pleural adhesions surrounding the upper lobe on all sides and thin fibrous adhesions over the entire surface of the lung. The mediastinum had little mobility.

Suction was applied by catheter (without proper regulation) while the thoracic wall was being closed. When the initial attempt to measure intrapleural pressure was made, the negative pressure was so great that the fluid in the manometer was

aspirated forcefully over into the long tube leading to the thorax. The catheter was opened immediately to allow the readjustment of intrapleural pressure to atmospheric level. The blood pressure at this time was 90 mm. of mercury systolic and 60 diastolic. The pulse rate was 100 per minute. Only a few minutes before, when the thorax was still open, the blood pressure had been 62/40. It had risen to 90/60 rather than fallen to that level.

At 1:45 P.M., while the patient was lying on his right side, the blood pressure was 100 mm. of mercury systolic and 60 diastolic. Pulse rate was 104 per minute. The intrapleural pressure was –24 cm. of water on inspiration and +8 cm. on expiration. At 1:48, while the patient was lying on his back in a horizontal position, the blood pressure was 90 mm. of mercury systolic and 60 diastolic; the pulse rate was 120 per minute. The intrapleural pressure was –14 cm. of water on inspiration and +10 cm. on expiration. At 1:50, while the patient was lying on his back with his head and shoulders elevated approximately 30 degrees, the blood pressure was 70 mm. of mercury systolic and 40 diastolic. The intrapleural pressure was –16 cm. of water on inspiration and +8 cm. on expiration.

From 2 to 2:15 P.M. bronchoscopic aspiration was performed. At 2:15 the blood pressure was 110 mm. of mercury systolic and 70 diastolic. At 2:25, while the patient was in bed in his own room, lying flat on his back, the blood pressure was 85/52 and the pulse rate was 120 per minute. At 2:45 P.M. the blood pressure was 88/50. At 2:50 he was placed in an oxygen tent. At 3:15 P.M. the blood pressure was 104/60 and the pulse rate was 120 per minute.

Fifteen hundred cubic centimeters of whole citrated blood were given during the course of the operation. A postoperative roentgenogram of the thorax showed the mediastinum in the midline.

CASE 3.—The patient was a man, aged 65 years. On May 8, 1944, right pneumonectomy was performed for extensive suppurative disease. There were many dense adhesions to the anterior parietal (intercostal) pleura and to the mediastinal pleura over the pericardium, but the mediastinum as a whole seemed of about normal mobility.

Immediately after closure of the thorax, intrapleural pressure was determined through a 24 F. rubber catheter. Gentle suction had been maintained through this catheter during closure. At 3:10 P.M., while the patient was lying on his left side, the blood pressure was 90 mm. of mercury systolic and 70 diastolic; the pulse rate was 128 per minute. The intrapleural pressure was –15 cm. of water on inspiration and –2 cm. on expiration. At 3:16, while the patient was lying on his back in a horizontal position, the blood pressure was 90 mm. of mercury systolic and 60 diastolic. The intrapleural pressure was –2 cm. of water on inspiration and +4 cm. on expiration. At 3:20, while the patient was on his back with his head and shoulders elevated approximately 30 degrees, the blood pressure was 85 mm. of mercury systolic and 60 diastolic; the pulse rate was 128 per minute. The intrapleural pressure was –8 cm. of water on inspiration and +6 cm. on expiration. At 3:21, while the patient was on his back in a horizontal position, the blood pressure was 85 mm. of mercury systolic and 60 diastolic; the pulse rate was 128 per minute.

From 3:22 to 3:32 P.M. bronchoscopic aspiration was performed. At 3:32, while the patient was lying on his back, immediately after bronchoscopy, the blood pressure was 94/60 and the pulse rate was 146 per minute.

At 3:45 P.M., when the patient was in bed in his hospital room, the blood pressure was 88/50 and the pulse rate was 120 per minute. He was placed in an oxygen tent almost immediately and during the next two hours the systolic blood pressure gradually increased to 105 mm. of mercury.

One thousand cubic centimeters of whole citrated blood were given during the operation. A thoracic roentgenogram made a few hours after the operation showed the mediastinum in the midline.

CASE 4.—The patient was a man, aged 48 years. On May 22, 1944, right pneumonectomy was performed for bronchogenic carcinoma. There was no pleural thickening. The pleural sac was free of adhesions; the mediastinum was mobile.

Measurements of intrapleural pressure were taken through a catheter, which had been used for maintenance of gentle suction during closure. At 2:28 P.M., while the patient was lying on his left side, the blood pressure was 100 mm. of mercury systolic and 60 diastolic. The pulse rate was 84 per minute. The intrapleural pressure was –12 cm. of water at the interrespiratory phase, immediately before the onset of inspiration. At 2:31, while the patient was lying on his back in a horizontal position, the blood pressure was 105 mm. of mercury systolic and 70 diastolic; the pulse rate was 84 per minute. The intrapleural pressure at the interrespiratory phase was +2 cm. of water. At 2:32, while the patient was on his back with his head and shoulders elevated approximately 30 degrees, the blood pressure was 105 mm. of mercury systolic and 70 diastolic; the pulse rate was 96 per minute. The intrathoracic pressure at the interrespiratory phase was –2 cm. of water. At 2:37, while the patient was on his back in a horizontal position, the blood pressure was 110 mm. of mercury systolic and 80 diastolic; the pulse rate was 96 per minute.

CASE 5.—The patient was a man, aged 61 years. On June 8, 1944, right pneumonectomy was performed for bronchogenic carcinoma. There were no pleural adhesions. The mediastinum was mobile.

Measurements of intrapleural pressure were taken through a catheter, which had been used for maintenance of gentle suction during closure. Throughout the operative procedure the systolic blood pressure varied from 120 to 90 mm. of mercury. At the completion of the procedure it was 110 mm. The pulse rate, which was initially 80, rose to 100 per minute. At 2:11 P.M., after the incision had been closed and while the patient was lying on his left side, the blood pressure was 120 mm. of mercury systolic and 70 diastolic; the pulse rate was 84 per minute.

At 2:15 P.M., while the patient was lying on his left side, the blood pressure and pulse rate were the same as at 2:11. The intrapleural pressure at the interrespiratory phase, immediately before onset of inspiration, was +4 cm. of water. At 2:19, while the patient was lying on his back in a horizontal position, the blood pressure was 110 mm. of mercury systolic and 60 diastolic; the pulse rate was 76 per minute. His intrapleural pressure at the interrespiratory phase was +12 cm. of water. At 2:20, while the patient was on his back with his head and shoulders elevated approximately 30 degrees, the blood pressure was 105 mm. of mercury systolic and 60 diastolic; the pulse rate was 76 per minute. The intrapleural pressure at the interrespiratory phase was +6 cm. of water. At 2:30, immediately after bronchoscopic aspiration, the patient's blood pressure was 110 mm. of mercury systolic and 60 diastolic; the pulse rate was 88 per minute. He was given 100 per cent oxygen by mask. At 2:40, immediately before the patient left the operating room, the blood pressure was the same as at 2:30.

At 3 P.M. the first determination of blood pressure after the patient had been transported on a litter to his room and placed in bed was made while he was in a horizontal dorsal position. The blood pressure was 80 mm. of mercury systolic and 60 diastolic. Almost immediately he was placed in an oxygen tent. Within one hour the blood pressure had risen to 120 mm. of mercury systolic and 80 diastolic. During this period the pulse rate varied from 60 to 80 per minute. A roentgenogram of the thorax showed the mediastinum in the midline. Twelve hundred cubic centimeters of whole citrated blood were given during the operation.

The concentration of hemoglobin was 14.0 Gm. per 100 c.c. of blood before the operation and 12.3 Gm. forty-eight hours after the operation.

CASE 6.—The patient was a woman, aged 52 years. On June 12, 1944, right pneumonectomy was performed for bronchogenic carcinoma. There were no pleural

adhesions. The mediastinum was mobile. During closure, negative pressure of 6 to 8 cm. of water was applied to the right pleural cavity through a rubber catheter, which was withdrawn immediately after closure.

There was a brief episode of brisk hemorrhage from a small rent in the wall of the pulmonary artery. The systolic blood pressure fell to 70 mm. of mercury at this time but rose rapidly to satisfactory levels when the bleeding was controlled. At 1:30 P.M., while the patient was lying on her left side at the completion of the operation, the blood pressure was 105 mm. of mercury systolic and 70 diastolic.

The patient's head and shoulders were elevated about 30 degrees during application of a dressing to the wound. At 1:35 P.M., immediately after this had been done and while the patient was lying horizontally on her back, the blood pressure was 85 mm. of mercury systolic and 60 diastolic. From 1:35 to 1:45 bronchoscopic aspiration was performed. At 1:45 the patient's blood pressure was 100 mm. of mercury systolic and 70 diastolic. From 1:45 to 1:48 she was given 100 per cent oxygen by mask. At 1:48 the blood pressure was 110 mm. of mercury systolic and 75 diastolic.

At 2 P.M. the first determination of blood pressure after the patient's arrival in her room was made. At this time the blood pressure was 90 mm. of mercury systolic and 50 diastolic; the pulse rate was 128 per minute. One hour after this the blood pressure had risen to 100 mm. of mercury systolic and 60 diastolic; the pulse rate had fallen to 104 per minute.

The patient's blood pressure and pulse rate remained stable during the next twenty-four hours. At the end of this time she was taken out of the oxygen tent* for one hour. During this interval she became quite pale, her nail beds became slightly cyanotic, and her blood pressure fell abruptly from 100/60 to 66/40. After her return to the oxygen tent the blood pressure rose to the previous level and her general condition improved promptly.

One thousand cubic centimeters of whole citrated blood were given during the operation. The concentration of hemoglobin was 12.3 Gm. per 100 c.c. of blood before operation and 12.1 Gm. forty-eight hours after operation.

CASE 7.—The patient was a woman, aged 31 years. On June 12, 1944, left pneumonectomy was performed for a large bronchial adenoma. Some pleural adhesions were present but the mediastinum was mobile.

The thorax was closed without suction by catheter in the pleural space during the procedure. A roentgenogram of the thorax taken immediately after the operation revealed a considerable shift of the heart and other mediastinal structures to the right. Two hundred cubic centimeters of air were removed from the left pleural cavity without visible change in the position of the mediastinum and without apparent influence on the blood pressure or pulse rate. Several hours later, with the aid of the pneumothorax apparatus, intrapleural pressure was found to be approximately +16 cm. of water immediately before the beginning of inspiration. After the withdrawal of about 700 c.c. of air the pressure was reduced to −2 or −4 cm. of water. Another roentgenogram of the thorax showed that the mediastinum had returned to the midline. This readjustment of intrapleural pressure occupied about five minutes. There was no change of blood pressure or pulse rate, though the respiratory rate immediately fell from 24 per minute to 16 per minute (Figs. 1 and 2).

CASE 8.—The patient was a boy, aged 16 years. Left pneumonectomy was performed for marked saccular and cystic bronchiectasis involving the entire lung. There were thin fibrous adhesions over the entire surface of the lung. The mediastinum appeared fairly mobile. During closure, suction through a temporary catheter was maintained.

*All patients routinely are placed in an oxygen tent when they are returned to their rooms after pneumonectomy.

The blood pressure had remained stable throughout the procedure (100 to 108 mm. of mercury systolic). The pulse rate had risen from 80 to 120 per minute. At 12:30 P.M., while the patient was lying on his right side at the completion of closure of the incision, the blood pressure was 115 mm. of mercury systolic and 70 diastolic; the pulse rate was 132 per minute. The patient's head and shoulders were elevated approximately 30 degrees during the application of a dressing. At 12:35, while he was horizontal on his back, the blood pressure and pulse rate were the same as at 12:30. From 12:35 to 12:47 bronchoscopic aspiration was performed. At 12:47 the patient's blood pressure was 95 mm. of mercury systolic and 60 diastolic; the pulse was rapid and irregular. He was given 100 per cent oxygen by mask. At 12:50 the blood pressure was 90 mm. of mercury systolic and 60 diastolic; the pulse rate was 126 per minute and the pulse was regular.

At 1 P.M. the first determination of blood pressure after the patient's arrival in his room was made. The systolic pressure was 50 mm. of mercury; the diastolic pressure could not be determined. The patient was placed in an oxygen tent within a few minutes. At 1:30 the blood pressure was 80 mm. of mercury systolic and 50 diastolic; the pulse was of good volume and his skin was warm and dry. At 9 P.M. the blood pressure was 90 mm. of mercury systolic and 55 diastolic. At 10 P.M. his blood pressure was 100 mm. of mercury systolic and 60 diastolic.

A roentgenogram of the thorax taken immediately after operation showed very slight shift of the mediastinum to the right. This was so slight that readjustment of the intrapleural pressure was thought unnecessary.

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TUBERCULOUS ABSCESS OF THE THYROID GLAND

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THERE is at the present time a considerable amount of literature on tuberculous infection of the thyroid gland, a condition, however, considered rare by numerous surgeons with extensive experience in thyroid surgery. Rokitansky[1] stated that tuberculosis of the thyroid gland was nonexistent, while Virchow[2] assumed it to be extremely rare. With the advent of thyroid surgery, case reports of tuberculosis of the thyroid gland began to appear in the literature, the diagnosis having been made in the majority of cases on the basis of extensive microscopic examinations of surgically removed goiters. All cases reported prior to 1913 were reviewed by Pollag,[3] who added cases of his own. In this country, Mosiman in 1917,[4] gave an excellent review of the literature at that time predominately European, and reported nine cases studied up to 1917 at the Crile Clinic. Coller and Huggins, in 1926,[5] added five more cases to a list of 105 apparently proved cases of tuberculosis of the thyroid gland. Twenty-one cases were reported from the Mayo Clinic in 1932 by Rankin and Graham,[6] together with 104 cases of surgically treated cases taken from the literature up to that time. Since then, case reports have been given by a number of clinicians, and at the present time the total number of recorded cases of tuberculosis of the thyroid gland is over 130. On reviewing these cases, however, it appears that there still is considerable confusion as to the criteria necessary to establish a definite diagnosis of tuberculosis of the thyroid gland. The diagnosis is questionable in a number of cases reported.

Because of the relatively infrequent involvement of the thyroid gland as compared with other organs in patients suffering from pulmonary tuberculosis, an immunity of the thyroid gland to the invasion of the tubercle bacillus was assumed by a number of clinicians. This is still the consensus of most investigators. Tuberculosis of the thyroid gland was produced experimentally by direct injection of suspensions of the bacilli into the thyroid artery by Torri[7] in dogs, and into the carotid artery by Roger and Garnier[8] in rabbits and guinea pigs. Shimodiara[9] was able to show that the spleen, kidney, and testis were more easily infected than the thyroid gland in rabbits. No conclusive experimental evidence is available to date to explain this apparent resistance of the thyroid gland to invasion by the tubercle bacilli.

The sclerotic changes of the thyroid gland associated with atrophy found in necropsy material of patients having died of chronic pulmonary tuberculosis were first described by Morin;[10] however, studies by Roger

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and Garnier[8] demonstrated that these changes in the thyroid gland were due to the toxemia of the tuberculous disease, rather than actual invasion of the gland by the tubercle bacilli. Kashiwamura[11] found similar sclerosing changes in the thyroids of patients having died of other chronic nontuberculous diseases.

In the overwhelming hematogenous dissemination of the tubercle bacillus in miliary tuberculosis, the thyroid, as do the other organs of the body, becomes the seat of tuberculous infection. However, since death occurs invariably in a short time, such infection of the thyroid does not present a clinical problem in diagnosis or treatment, and only postmortem material is available for study. Such cases have been reported in the literature.[12-14]

The diagnosis of tuberculosis of the thyroid gland in the majority of cases reported in the literature was based on routine histologic examination of thyroid tissue removed at thyroidectomy. In only a few patients was such a diagnosis suspected preoperatively and the diagnosis in the largest number was goiter, both toxic and nontoxic, thyroiditis, or carcinoma. The age of these patients usually fell in the fourth and fifth decades, and examinations, particularly of their lungs, failed to reveal any evidence of pulmonary tuberculosis or tuberculous infection anywhere else in the body. At the time of operation there was, as a rule, diffuse enlargement of the thyroid with nodule formation, and the diagnosis during operation was essentially that of the preoperative diagnosis. The postoperative course was uneventful and in all these cases there was primary healing of the wound. The patients were discharged and the diagnosis of tuberculosis of the gland was made days later on the basis of the histologic changes found in the microscopic sections. Thus, at the time of the true diagnosis of the condition, no fresh tissue was available, and culture or guinea pig inoculation could not be performed. An attempt was made to demonstrate the tubercle bacilli in the fixed tissues, dissolving the tissue in antiformin, and after concentration, staining the material by the usual Ziehl-Neelsen method. Using this approach, Ruppaner[15] was able to demonstrate tubercle bacilli in one of his three cases, while Hedinger[16] reported having found tubercle bacilli in six of his seven cases. In this country, Mosiman[4] used the same method but was not able to demonstrate the tubercle bacilli, although he found acid-fast particles which could be mistaken for tubercle bacilli; however, they did not exhibit the typical morphology. Others who attempted to demonstrate tubercle bacilli by staining of fixed tissues or extracting the tissue with the antiformin method, but were unable to do so, were Dinsmore[17] in twenty-one cases, Rankin and Graham[6] in twenty-one cases, and Jaffe[18] in four cases. The diagnosis of tuberculosis of the thyroid gland in the majority of cases, then, was based on histologic findings and interpretation, rather than by demonstrating the tubercle bacilli.

The histologic picture reported as being diagnostic of tuberculosis of the thyroid gland was the finding of both interfollicular and intrafollicular giant cells resembling Langhans cells, lymphocyte and round-cell infiltration with fibrosis. Jaffe[18] was the first to point out that the tubercle-like structures in these glands were the results of noninfectious focal involutional changes of newly formed and old follicles. Rienhoff[19] and Seed[20] demanded the demonstration of the tubercle bacilli before a diagnosis of tuberculous infection of the thyroid gland could be made with certainty.

Direct extension of a tuberculous process from cervical lymph glands,[21] muscles,[2] and trachea and larynx[8] to the thyroid gland has been reported. Such cases are not of particular surgical interest, the pathology being demonstrated in post-mortem material. The diagnosis of tuberculosis of the thyroid in these cases was based predominantly on finding extensive tuberculosis in the lungs and other organs.

There have appeared in the literature reports of sixteen cases of tuberculous abscesses of the thyroid gland. These abscesses, as a rule, developed in normal thyroid glands and clinically were characterized by a slowly increasing fluctuant swelling in one lobe or isthmus. The treatment consisted of incision and drainage, and was followed by delayed healing of the wound or sinus formation. The aspirated pus, when smeared or cultured, contained tubercle bacilli, and the granulation tissue removed was composed of tuberculous granulation. Tuberculous abscess of the thyroid gland was occasionally associated with tuberculosis elsewhere in the body, although the primary source of the infection could not be found in all cases. The thyroid function was not altered prior to and following incision and drainage of the abscess.

CASE REPORTS FROM THE LITERATURE

In 1894, Schwartz[22] reported the first surgically treated case of tuberculous abscess of the thyroid. His patient was a 30-year-old man with a swelling of the right lobe of the thyroid. The mass moved on swallowing and the larynx was deviated to the left. On incision pus was obtained which was sterile on the usual culture but acid-fast bacilli were demonstrated by guinea pig inoculation. The incision healed. No other foci of tuberculosis were reported.

Ivanoff[23] reported a probable case of tuberculous abscess in a 23-year-old man. A swelling had appeared three months previously over the right thyroid gland, which became fluctuant, and on incision pus was obtained. No attempt was made to find the tubercle bacilli, but since the patient died of miliary tuberculosis following the development of tuberculosis of the left ileum and knee ten months later, Ivanoff made a diagnosis of tuberculosis of the thyroid gland.

In a boy 2 years of age, Clairmont,[24] in 1902, found a walnut-sized tumor in the region of the isthmus which, because of obstructive symptoms, was incised and found to contain pus. Acid-fast bacilli were not

demonstrated; however, the tissue removed showed tuberculous granulation tissue. The wound healed with difficulty and a draining sinus persisted for months. There was no evidence of tuberculosis elsewhere.

In a 42-year-old man with pulmonary tuberculosis limited to the left upper lobe a fluctuant mass was found by Pupovac[25] and on incision pus was obtained from an abscess of the isthmus. The diagnosis was made on the basis of histologic examination. The wound healed promptly. There was no evidence of hyperthyroidism.

Corner,[26] in 1904, reported on a 9-year-old girl who had a fluctuant mass in the right thyroid with a hard left thyroid lobe. After incision and drainage the wound did not close and the patient died seven months later of tuberculous meningitis and miliary tuberculosis.

The diagnosis of tuberculosis of the thyroid gland in the case of Ledidard[27] may be questioned. A male of 21 years had a fluctuant mass in the right thyroid gland. On puncture, pus was aspirated which was sterile, but contained numerous giant cells. There was complete healing of the puncture wound and no recurrence of the tumor.

v.Schiller[28] was the first to make a diagnosis of tuberculosis of the thyroid gland during the operation on a patient with a cystic tumor of the left thyroid gland. This 17-year-old male had tuberculosis of the lungs. The pus obtained with incision of the cyst contained tubercle bacilli and was rich in iodine.

Lenormant[29] reported a case, in 1908, of a man 40 years of age, with tuberculous osteomyelitis of the trochanter. There was a fluctuant swelling of the isthmus of the thyroid, which on incision contained pus. A sinus persisted after the operation.

In 1910, Halstead[30] reported a case of tuberculosis of the thyroid gland in a 28-year-old woman. A swelling in the thyroid region ruptured spontaneously with the discharge of a large amount of pus. The lining of the abscess was composed of tuberculous granulation tissue. Acid-fast bacilli were not demonstrated.

Pollag,[3] in 1913, reported two cases of tuberculosis of the thyroid with abscess formation in which tubercle bacilli were demonstrated on smear. The first case was that of a 59-year-old white woman who had had a goiter since childhood. On examination she was found to have tuberculosis of the right knee and also of the lungs. A goiter was present; however, a fluctuant mass in the isthmus was incised and pus obtained. The wound healed in six weeks. The second case was that of a 74-year-old woman who presented a fluctuant swelling of the right thyroid lobe. There was no evidence of tuberculosis elsewhere. On incision, grayish-brown pus was obtained. The wound did not heal for five months.

Jean[31] made a diagnosis of tuberculosis of the thyroid gland in a man of 42 years, who presented a cystic swelling of the thyroid. On incision, pus was obtained but no acid-fast bacilli were demonstrated. The wound healed promptly. There was no tuberculosis found anywhere else in the body.

In 1929, Jones[32] reported a man 64 years of age, who had a tumor the size of a walnut in the region of the isthmus. There were no symptoms of hyperthyroidism. A thyroidectomy was done and the tumor was found to be an abscess, the pus of which contained tubercle bacilli on guinea pig inoculation. The wound did not heal and ultraviolet radiation was used. The patient did not return for follow-up observations.

A patient of 26 years, with pulmonary tuberculosis and all the symptoms of a toxic goiter, was operated upon by Keynes in 1938.[33] The right thyroid contained an abscess and the pus on smear was positive for acid-fast bacilli. The abscess wall showed tuberculous granulation tissue. The patient made an uneventful recovery from the thyroidectomy.

In 1941, two cases of abscesses of the thyroid gland were reported by Hare and Simpson[34] from the Lahey Clinic. A woman of 65 years had a swelling of the left lobe of the thyroid which on incision drained pus containing gram-positive cocci but no growth on culture. The wound healed with difficulty and x-ray was used, with healing of the wound in eleven months. The second patient was a woman 30 years of age. There was swelling in the region of the isthmus. No toxic symptoms were present. Pus was drained from an abscess cavity, the wall of which consisted of tuberculous granulation tissue. No acid-fast bacilli were demonstrated. X-ray treatment was used to aid in the healing of the wound, sinuses persisting for eight months.

AUTHORS' CASE REPORT

M. J. (No. 424066), a 21-year-old Negro student nurse, was admitted to the Starling-Loving University Hospital, June 14, 1942, complaining of a painful swelling in her neck and of afternoon fever. Four months prior to admission she developed a small swelling on the right side of the neck which gradually increased in size and was associated with difficulty in swallowing. In March, 1942, routine x-ray examination had shown what was interpreted as an early tuberculous lesion of the left lung and the patient was advised to discontinue her activity and go on absolute bed rest. She had slight afternoon temperature elevation, but no other symptoms referable to the chest. There had been no cough and no weight loss. There was no family history of tuberculosis.

The patient was a well-developed Negro woman. The pulse on admission was 96, with a temperature of 99.4° F. The blood pressure was 138/86 mm. There was no lymphadenopathy. There was a round, fluctuant enlargement of the lower portion of the right thyroid lobe, while the isthmus and left lobes were normal in size and consistency. The cystic swelling of the thyroid did not transilluminate and it was freely movable, not attached to the skin or muscles. Examination of the chest revealed normal breath sounds and the heart was not enlarged.

The red blood count was 3,860,000, with a hemoglobin of 11.5 Gm. The white blood count was 6,200, with 78 per cent neutrophiles, 18 per cent lymphocytes, 3 per cent monocytes, and 2 per cent eosinophiles. The sedimentation rate was 2.0 mm. per minute. The basal metabolic rate was minus 21 per cent and the blood iodine 5.1 micrograms per cent. X-ray films of the chest showed an area of increased density in the second interspace on the left side, which had all the appearance of a minimal tuberculous lesion. There was no excavation. The trachea

just below the larynx was deviated to the left. The urine was normal. Sputum examination did not demonstrate acid-fast bacilli.

A diagnosis of hemorrhagic cyst of the thyroid and pulmonary tuberculosis, minimal, apparently active, was made. Using cyclopropane and oxygen anesthesia, the thyroid was exposed through a low collar incision. The left lobe and isthmus of the thyroid were normal in size and appearance. The upper portion of the right lobe was normal while the lower portion was adherent to the adjacent structures, and in trying to mobilize it the capsule was broken into with the escape of a considerable amount of greenish-gray thick pus. Exploration with the finger revealed a large cavity with ragged wall which had replaced the lower portion of the left lobe of the thyroid. The wound was closed with drainage. The pus was sterile on usual culture and no organisms were seen. Guinea pig inoculation was done. The guinea pig died six weeks later of tuberculosis, acid-fast rods being found in the spleen, liver, and site of injection.

The wound did not heal and the patient was discharged to her home on absolute bed rest. Dr. Ralph Holmes of Chillicothe, Ohio, gave her eight x-ray treatments of 100 r. each at three-day intervals, to aid in the healing of the wound. Examination Nov. 14, 1942, revealed that the wound was closed, and x-ray examination of the chest showed the tuberculous lesion to be smaller. The patient was allowed gradual increase in her activity and when last examined, the lesion in her lungs had disappeared except for a small area of fibrosis in the second interspace. The patient returned to work in December, 1943, at which time there was no evidence of activity in her lungs. The basal metabolism, Feb. 1, 1944, was minus 10 and x-ray examination of the chest was reported as being normal.

DISCUSSION

Our case and the majority of those collected from the literature present the criteria necessary for the absolute diagnosis of tuberculosis of the thyroid gland, as advocated by Seed.[20] He states that a diagnosis of tuberculosis of the thyroid gland can be made with certainty only when tubercle bacilli are found, when there is definite necrosis or abscess formation, and when there is an etiologic focus in the body outside the thyroid gland. In the seventeen cases presented in this report, tubercle bacilli were demonstrated in seven. In Pollag's two cases the tubercle bacilli were found on direct smear, while v.Schiller and Keynes obtained positive cultures. Schwartz, Jones, and we demonstrated the acid-fast bacilli by guinea pig inoculation. In the other ten cases, the tubercle bacilli were not demonstrated; however, there is no report of an attempt having been made to find the bacilli. Apparently no smear was made, nor was there guinea pig inoculation or culture. However, the appearance of the pus and the absence of other organisms, with the histologic appearance of the removed tissue, was assumed sufficient proof to make a diagnosis of tuberculous abscess of the thyroid gland.

Four patients with tuberculous abscess of the thyroid gland also had tuberculosis of the lungs, which in all probability was the source of the infection, which, however, was not of sufficient severity to cause death. Only two of these patients, the one reported by Keynes and our case, were receiving treatment for the pulmonary tuberculosis. Four patients had tuberculous osteomyelitis and of these one died of miliary

tuberculosis several months after the drainage of the thyroid abscess. Corner's patient had cervical lymphadenopathy and died seven months later of tuberculous meningitis and miliary tuberculosis. No other foci of tuberculous infection were demonstrated in eight of the reported cases. Since routine x-ray examination of the chest was not used and no tuberculin test was made, these patients may be assumed to have had a primary focus which was not found on routine examination.

The treatment of tuberculous abscess of the thyroid consisted of incision and drainage in thirteen of the cases; one abscess ruptured and drained spontaneously, while a thyroidectomy was done in two cases. In the largest number of patients the wound healing was characterized by delayed wound healing and sinus formation lasting up to one year. Ultraviolet radiation was used by Jones and x-ray treatment by Hare and Simpson and us in an attempt to accelerate delayed wound healing. In six patients the wound healed by primary intention.

Definite goiter was present in three patients, one of which was toxic. The latter had a thyroidectomy with recovery. The amount of thyroid tissue destroyed by the tuberculous infection was not sufficient in these patients to cause postoperative hypothyroidism.

SUMMARY

A case of tuberculous abscess of the thyroid gland is reported, together with a brief outline of sixteen cases selected from the literature. Tuberculous abscess of the thyroid was equally distributed between males and females and the ages varied from 7 to 74 years. Three patients had definite goiters, of which one presented toxic symptoms. The clinical picture of tuberculous abscess was that of a cystic swelling of the thyroid gland of several months' duration, which on incision was found to contain thick pus, positive for acid-fast bacilli on smear, culture, or guinea pig inoculation. The tissue removed showed tuberculous granulation. There was delayed healing of the wound in the majority of cases. In no patient was postoperative myxedema demonstrated. A focus of tuberculous infection outside the thyroid gland was demonstrated in nine patients. Two patients died of miliary tuberculosis, while the others made uneventful recoveries.

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INTRASTERNAL INFUSIONS AND TRANSFUSIONS

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IN MILITARY and civilian medicine the treatment of shock is of paramount importance. Whether the condition is brought about by wounds, hemorrhage, or burns, the immediate physiologic results are the same, and there is an urgent need for the parenteral administration of fluids. These fluids can usually be given by vein, but in some instances the veins are collapsed, thrombosed, or absent, or the vein areas are obliterated by wounds or burns.

The bone marrow offers a ready channel for the administration of fluids in these cases, as first described by Tocantins.[1]

The present report is a study of the administration of various fluid mediums through the sternal marrow. The work was done on routine patients in the general wards of a large city hospital.

The technique of sternal puncture and infusion is very simple. The needle used is 1¼ inch, 16 gauge Luer type with a stylet. The site of puncture is anywhere in the midline of the sternum but preferably in the region of the second or third interspaces.

A small spot of iodine is painted over the puncture site, novocain is injected into the skin, subcutaneous tissues, and periosteum, and the sternal needle is tapped into the marrow cavity by means of a small mallet. The mallet technique is superior to pushing the needle because perfect control is exercised and the danger of perforating the sternum and injuring the heart and great vessels is eliminated.

The stylet is removed and suction applied with a 10 c.c. syringe. Aspiration of bloody fluid proves whether the needle is in the marrow cavity. The infusion is then begun in the usual way.

Our series consists of thirty-five infusions of all types of fluids. The experimental data are given in Table I.

As seen from Table I, all fluids except blood ran in easily. That includes saline, glucose, sulfonamide drugs, insulin, serum, and plasma. Difficulties were encountered with blood and that in varying dilutions. This is hard to explain except on the basis of the size of the particulate matter in the blood in relation to the histologic structure of the marrow.

The internal structure of the sternum consists of spongy bone in which there are masses of cells traversed by thin-walled sinuses.

At sternal puncture the needle is driven through the outer body plate into the cellular mass in the interior of the sternum. It is plain to see how fluids not containing particular matter could traverse the cell masses and get into the capillaries much more easily than fluids con-

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TABLE I

PUNCTURE NUMBER	AGE	SEX	DIAGNOSIS	SOLUTION	REMARKS
1	77	M	Virus pneumonia	5% Dextrose in saline	2000 c.c. ran in well in 4 hr.
2	86	M	Virus pneumonia	5% Dextrose in saline	1000 c.c. ran in well in 2 hr.
3	63	M	Myelogenous leucemia & virus pneumonia	Whole blood citrated	Unsuccessful; sternal needle inserted, marrow withdraws easily but solution would not run in
4	70	F	Gastric carcinoma	5% Glucose in saline	3000 c.c. ran in well in 7 hr.
5	71	F	Carcinoma of head of pancreas	Whole blood citrated	Unsuccessful; would not run in by gravity; 100 c.c. given by syringes with difficulty
6	73	F	Carcinoma of stomach	5% Glucose in saline	4000 c.c. ran in easily in 10 hr.
7	65	F	Diabetes mellitus	5% Glucose in saline	2000 c.c. ran in easily
8	80	F	Carcinoma of stomach	5% Glucose in saline	2000 c.c. ran in easily
9	65	F	Diabetes mellitus & hemiplegia	5% Glucose in saline	3000 c.c. ran in easily
10	73	F	Metastatic carcinoma	5% Glucose in saline	3000 c.c. ran in easily
11	73	F	Carcinoma of stomach	5% Glucose in saline; sulfathiazole, 5 Gm.; nicotinic acid, 100 mg.; Vit. B, 50 mg.; Vit. C, 200 mg.	3000 c.c. ran in easily
12	73	F	Metastatic carcinoma of stomach	5% Glucose in saline	2000 c.c. ran in easily
13	59	M	Carcinoma of rectum	5% Glucose in saline	2000 c.c. ran in easily
14	43	M	Carcinoma of rectum	Saline, 100 c.c.; whole blood citrated, 23 c.c.	Saline ran in well; blood added, infusion stopped; needle washed out with saline, tried everything to get running again; unsuccessful
				Saline 100 c.c.; citrated blood 100 c.c.	Needle reinserted 4 hr. later; blood diluted 50:50 with saline; ran in with great difficulty
				5% Dextrose in saline	3000 c.c. ran in well through same needle following unsuccessful administration of blood
15	43	F	Shock, temp. 107° F.	5% Glucose in distilled water plus Hartman's solution	1000 c.c. ran in slowly except when respirations were deep and even, then it ran in quite rapidly
16	40	F	Anemia	Whole blood citrated	100 c.c. saline ran in; stopped when blood was added; unsuccessful
17	44	F	Anemia	Saline, whole blood citrated	100 c.c. saline ran in well; stopped when blood was added; injected some with syringes with great difficulty

TABLE I—CONT'D

PUNCTURE NUMBER	AGE	SEX	DIAGNOSIS	SOLUTION	REMARKS
18	70	M	Anemia, 20% Hb.	Saline with whole blood citrated	100 c.c. saline ran in then stopped when blood was added
19	70	M	Anemia	Whole blood citrated	Tried to inject forcibly with syringes; had to discontinue
20	36	M	Anemia	5% Glucose in saline	1000 c.c. ran in slowly
21	73	F	Carcinoma of esophagus	5% Glucose in saline	4000 c.c. ran in very well
22	64	F	Uremia	5% Glucose in saline; Sod. sulfathiazole, 7 Gm.	2000 c.c. ran in very well
23	73	F	Carcinoma of esophagus	5% Glucose in saline	3000 c.c. ran in very well
24	64	F	Pneumococcus meningitis	5% Glucose in saline; sulfadiazine, 2 Gm.; antipneumonia serum, 100,000 U. type 8	3000 c.c. ran in very well
25	32	F	Postoperative shock	Saline and whole citrated blood	500 c.c. saline ran in well; 100 c.c. blood ran in slowly, then stopped
26	64	F	Pneumococcus meningitis	5% Glucose in saline	3000 c.c. ran in very well
27	38	F	Pneumonia	Whole blood citrated	Diluted 50:50 with saline; ran in too slowly, discontinued
28	70	F	Carcinoma of stomach	Whole blood citrated	100 c.c. forced in with syringes; discontinued as unsuccessful
29	60	F	Diverticulitis of colon	5% Glucose in saline	2000 c.c. ran in very well
30	60	F	Hemorrhage	Whole blood citrated	50 c.c. forced in with syringes; discontinued
31	72	M	Malnutrition, hypoproteinemia	Plasma	Ran in very well
32	48	M	Cirrhosis of liver	Plasma	Ran in very well
33	49	M	Nephrotic syndrome	Plasma	Ran in very well
34	75	F	Anemia malignancy	Plasma	Ran in very well
35	62	F	Postoperative shock	Plasma	Ran in very well

taining cells, which would be caught by the sticky masses of primitive cells in the marrow and cause a blockage.

To prove that this was the case, saline was run into the sternum after the blood blockage had occurred, and it went in easily. To show that there was nothing wrong with the blood which would not go into the sternum, it was run into the same patient's vein.

In spite of the disappointment with blood, the sternal route of administering fluids and plasma is very satisfactory.

An interesting variation of technique has been developed in patients who have collapsed veins and need blood in addition to fluids. In these cases the fluids are first given by sternum until the circulation volume is increased to such a point that the veins again stand out. The blood transfusion is then given through the veins.

CONCLUSIONS

1. Intrasternal infusions of saline, glucose, and plasma are practical.
2. Intrasternal transfusions of blood were unsuccessful.
3. The sternal route is of great value in the treatment of shock.

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TRENCH FOOT

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DURING the past winter many of our soldiers suffered from trench foot, a condition manifested by pain and swelling of the feet following exposure of the parts to wet and cold, usually in a trench or foxhole. The observations that follow are based on the study of 125 of these patients at a general hospital.

Trench foot, frostbite, immersion foot, shelter foot, and chilblain are closely related.[1] The main difference among these conditions is the manner of occurrence. The majority of our patients were suffering from trench foot, but there were two with immersion foot. The signs, symptoms, and course of the latter could not be distinguished from those of the patients with trench foot.

PREDISPOSING FACTORS

One or any combination of several factors seemed to produce the same signs and symptoms. These factors are as follows:

1. *Wet and cold.*—These men were exposed to the cold and wet of Italy. The average time of continuous exposure before reporting to sick call was eleven days. The extremes were two and forty days. The patients did not know exactly the temperature of the weather but they agreed that it was not extremely cold. They thought that the thermometer remained well above freezing during the day but there was some ice formation at night. Greene[2] has noted that trench foot was less common at extremely low temperatures than when the temperature was a little above freezing and the ground wet and muddy. Similarly, dry cold weather is better tolerated than is moist cold. Eight degrees Fahrenheit or lower is necessary to produce severe frostbite.

2. *Dependent Position of Feet and Circulatory Stasis.*—Most of these soldiers were in foxholes for long periods at a time, consequently their feet were in a dependent position day and night. Greene[2] has observed that people who developed the condition known as shelter foot, characterized by swelling of the feet, spent days and nights in the air raid shelters of London. During this time their feet were in the dependent position almost constantly and pressure was exerted on the popliteal fossae. Pressure might be an etiologic factor, as was suggested by the observation that those who sat with one leg crossed over the other developed swelling in the lower leg. A similar observation was made by us in some of our patients who had the disease worse in one foot

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than in the other. One reason that we could offer, in most cases, to explain this was their statement as to the position they assumed in the foxhole.

3. *Change of Socks.*—A better idea of continuous exposure is offered by the fact that the average time these patients went without changing their socks was seven days. The extremes were one and twenty-five days. The men said their socks were usually wet. All the patients were wearing light or heavy wool socks during the time of their exposure. Thirty per cent of the patients wore either combat shoes or galoshes.

4. *Previous Attacks.*—In this group of patients 8 per cent had had previous attacks of frostbite or trench foot, but in only three had these occurred during World War II. The others had had trouble five to fifteen years previously. Only one of these recurrent cases was severe.

5. *Previous Foot Injury.*—Eleven per cent had had previous foot injury but only in two patients was this factor thought significant. One of these developed gangrene in one foot (one year before, he had broken that foot and it still became swollen after long walks) but had no trouble with the other foot. The second patient was the only soldier in his company affected. He stated that the others had the same exposure. Two years previously he had had crushing injuries to both feet.

6. *Ringworm Infection.*—Fifteen per cent had epidermophytosis of their feet recently. These figures are about the usual incidence for all soldiers.

7. *General Condition.*—Seventy-five per cent of the patients were in good or excellent general condition when seen by us. The others were in fair condition as evidenced by their statements of an average loss of only ten pounds of weight since the beginning of exposure. We were unable to determine the presence of any specific vitamin deficiency. Kappert[3] thinks there is a relationship between vitamin B deficiency and the development of frostbite but Stephenson and co-workers[4] disagree. He notes that the swelling associated with vitamin deficiencies is usually not accompanied by pain, which symptom is the most common complaint of sufferers with trench foot.

SIGNS AND SYMPTOMS

The symptoms are well known. After exposure to wet and cold for a period varying from a few hours to several days, the man complains of painful and swollen feet. Pain was often the first symptom, but in many cases swelling of the feet and inability to get the shoes on after a change of socks was noted. Indeed, 8 per cent of the patients did not know they had trench foot until they were sent to an aid station for a wound or sickness. After becoming warm their feet became swollen and painful.

Other symptoms were numbness and burning. Most patients complained of hot feet and would ask that all covering be removed from their feet. A trench foot ward presents an unusual sight in January.

The feet of all patients are protruding from neatly turned back covers. Only 15 per cent stated that at no time during the course of their disease did they feel better with their feet in the cool air.

Sweating and coldness were late symptoms which did not appear during the early part of the disease. At least three of the patients who were returned to duty complained of no late symptoms except profuse sweating of the feet.

All of the patients gave a history of having had swelling of the feet at some time during the first part of their illness. Sixty per cent had edema which persisted for more than three weeks. Some degree of pinking or ecchymosis accompanied this edema in most instances.

In 25 per cent of all the patients, pulsation in the dorsal pedal artery, posterior tibial artery, or both was absent. We were unable to determine a relationship between such finding and the severity of the disease, although the pulsations did seem stronger in the hot feet and weaker in the cold feet. Pulsations of these vessels are not always palpable in normal feet.

Most of our patients were seen one to two weeks after the beginning of hospitalization, therefore in many instances the first formation of blisters could not be observed. When these were present on arrival in this hospital, the vesicles contained either a clear or a sanguineous fluid and were similar in appearance to the descriptions of those occurring in high altitudes frostbite as given by Davis and others.[5]

In 9 per cent of our patients the degree of vesiculation was severe and in these cases the outcome was like that described by Davis and associates.[5] The skin dried, turned black, and dropped off, sometimes en masse. The affected toes were finally covered with thin, smooth, shiny, tender skin.

Six per cent of the patients developed a type of dry reaction which was similar to that seen in high altitude fliers. The tissues were shriveled, desiccated, and black. The affected part of the toe or toes would drop off, usually within six to eight weeks. Those patients who presented vesicle formation and persistent swelling of the feet and whose temperature varied frequently seemed to suffer more than those who had loss of toes.

SKIN TEMPERATURE

During their hospital stay, readings of skin temperature, usually the knee, shin, foot, and toe, were taken with an accurately calibrated skin thermometer. Often these readings were repeated on one or more occasions. Since it was not feasible to use a constant temperature room, an attempt was made to take readings when the room temperature was between 62 and 70° F. After the legs of the patient had been exposed for one-half hour, the usual "normal" temperature on the back of the foot varied from 70 to 80° F. and on the ball of the big toe from 60 to 70° F.

The patients of our group were seen usually from one to four weeks after the onset of their trouble and at this time the feet with the most damage were the warmest. Frequently the temperature of the injured foot and toe would be the same, contrary to the normal, and often above 85 or 90° F. Palpation of the feet would frequently lead to a statement of the relative degree of injury between the two feet because of a temperature difference. Where the trench foot was mild in degree, the skin temperature of the feet and toes often dropped to "normal" in one or two weeks. Usually the temperature was increased in the swollen feet; however, this was not true in some cases with swelling three and four months after injury. When damage was severe and gangrene of the toes was present, the temperature of the affected foot and base of the toes remained elevated almost without exception until after separation of dead tissue.

While skin temperature recordings were an aid in judging the condition and early course in the disease, they were of little value in the late cases in which the patients complained of sensitivity to cold. The skin temperature response of five such patients who had moderately severe trench foot of from seventeen to forty days' duration was studied simultaneously with five normal controls. These men were on beds with their legs exposed and were not allowed to move about. Skin temperature readings were made every hour for seven hours. The temperature of the room was 62° F. at the start and 68° F. at the end of the test period. The temperatures of the exposed legs, feet, and toes reached their lowest point about three hours after exposure, and the curves of the drop in the trench foot cases showed no significant variation from that of the normal. In other words, the response of the arterial sympathetics on exposure to temperatures of 62 to 68° F. was not different in "recovering" trench foot and in normal cases. Whether these damaged feet respond differently to lower temperatures, we did not observe.

PATHOLOGIC AND LABORATORY OBSERVATIONS

In an attempt to find a pathologic basis for the persistent symptoms in some patients without gangrene, skin biopsies were taken from one foot in three patients and from both feet in a fourth, about ninety days after onset of symptoms. All complained of persistent swelling, pain on walking, and occasional bluish discoloration. Control tissue was taken (post mortem) from four patients who had normal feet. All tissue was removed from the dorsum of the foot and consisted of full thickness of skin, with varying amounts of subcutaneous tissue. Sections were stained with hematoxylin eosin and with Mallory's aniline blue stain for collagen.

As White and Warren[6] have observed, the outstanding change in the tissues was an increased formation of collagen. This was most marked in the papillary layer of the dermis and surrounding the small blood

vessels in the dermis as a whole. The vessels, particularly of the papillae, were more numerous and appeared dilated, suggesting a more active capillary circulation. These vessels showed no evidence of thrombosis or constriction by the surrounding fibrous tissue. There was slight to moderate perivascular round-cell infiltration in all sections, which was not observed in the control tissues. A small amount of collagen formation was present diffusely throughout the dermis, although this layer showed no increase in thickness over that seen in the controls.

The epidermis was essentially normal in all sections. There was no significant smoothing of the rete pegs and the basal layer did not show the irregularity and indistinct arrangement noted by others.

Histologic determination of the presence of edema was uncertain. In the one patient in whom both feet were biopsied, it was not possible to determine by section which was the clinically edematous foot. Both feet had been edematous, but at the time of biopsy only one was swollen.

There was neither significant pathologic change in nerves nor marked fibrosis surrounding nerve trunks. The skin appendages were normal in number in three sections and slightly decreased in the other two. Histologic examination was made on gangrenous tissue removed from one patient, but this showed no changes not entirely characteristic of any dry gangrene, regardless of cause.

Sedimentation rates by the Westergren method and the presence of cold agglutinations were determined routinely in most patients. In only one patient was the sedimentation rate increased (40 mm.). In all others the rates covered a range between 0.5 and 3 mm. per hour. Cold agglutination was determined on saline washed red cells at a temperature of approximately 5° C. Since the determination of agglutinin titer was not practical, the results were recorded in terms of completeness of agglutination, 3 and 4 plus being those in which 75 to 100 per cent of the cells were clumped. Readings on twenty-two normal controls were made under the same conditions. Thirteen per cent of eighty-four patients with trench foot and 4.5 per cent of the control group showed 3 or 4 plus agglutination. If the basis for significant agglutination is broadened to include those patients showing 50 per cent of cells clumped, then 35.6 per cent of patients with trench foot and 22.6 per cent of the controls had agglutination of 2 plus or more. There was poor correlation between these findings and the degree of edema in nongangrenous feet, however, all patients who had gangrene and on whom determinations were made showed positive agglutination. The significance of these findings is nevertheless uncertain.

TREATMENT

The treatment employed in this hospital was simple. The patients were grouped for treatment purposes into mild, moderate, and severe types. Those with a mild type were kept in bed three to fourteen

days. The feet were slightly elevated if there was any pain. A modified type Buerger's exercise was begun when the pain and swelling had diminished. As soon as possible the patients with no signs were encouraged to walk, first without shoes and later with their regular shoes. When they were able to walk one-half mile and soak their feet alternately in hot and cold water without return of symptoms they were ready for discharge to duty or to a convalescent hospital, depending on the toughness of their feet.

The same plan was followed with the group of patients who were moderately affected. The rest period was longer and the exercise more graduated. They were given the same tests before being returned to duty.

All groups were given multivitamin capsules and large doses of vitamin B as recommended by Kuppert.[3]

Patients of the first two groups recovered without residual signs and symptoms but, as will be seen, it was necessary to hospitalize some patients for three months or longer before returning them to duty.

The treatment of the severe type was more of a problem. Twenty per cent were in this group. Blebs were opened under sterile precaution and sulfanilamide crystals used freely. Dry dressings were always applied. Necrotic tissue was cut off or washed away with saline solution at room temperature. The feet with gangrenous areas were treated the same way. There was no severe infection in any of these patients. None had a temperature of more than 100° F. at any time during their course in the hospital.

Brambel and Loker[7] have suggested the use of dicoumarin to check the spread of gangrene from frostbite. None was used in this series; since we have not found the gangrene progressive, we cannot see the value of either decoumarin or heparin.

The gangrenous parts were allowed to drop off and no skin grafting was necessary. In two patients it was necessary to cut off the protruding ends of bones of the toes. None of the patients with bleb formation received additional tetanus toxoid or antitoxin. None developed tetanus although tetanus has been reported following frostbite.

Pain, and in some instances swelling, was relieved by cooling. Usually exposure to the cold air of December and January of North Africa was sufficient. Davis and others[5] have found that the results obtained in the treatment of cases of frostbite at room temperature were as good or better than that of those treated by continued cooling. Ungley[8] and Greene[9] have reported good results using a dry refrigerator. Most writers on this subject advocate cooling by one method or another. Six of our patients whose pain was not relieved with ordinary cooling were improved by the use of ice bags. Two patients who were seen early in our experience were interesting. They had only slightly swollen feet but they complained of severe pain. When they were not

relieved of their pain by ordinary cooling they were given buckets of ice water and allowed to cool their feet. They received immediate relief of the pain but after several treatments the feet became swollen and blue. This form of therapy was discontinued. For those with more intense pain and severe swelling we now use dry cold in the form of protected ice bags and we have had no increase in the edema since the dry cold was used.

We have had little experience with sympathetic block in the treatment of this condition. It was tried without benefit on three patients with persistent pain and swelling of the feet. One patient said his feet hurt more. The only indications we can see for sympathetic block or sympathectomy would be, as Patterson[11] has described, for feet that remained cold with absent pulsations of the large arteries of the feet or possibly in that group of patients left with an unstable vasomotor system in the feet. However, these people feel better when their feet are cold than when they are hot. We would recommend careful study before performing a sympathectomy.

RESULTS

Trench foot, even in the milder cases, proved to be disabling. Those patients who were able to return to duty were hospitalized for an average of sixty-one days, with the extremes at 30 and 115 days. Eighty-two per cent (counting those who were referred to a convalescent hospital) were able to return to duty. Eighteen per cent were returned to the Zone of the Interior. Of this number three had a peculiar condition manifested by one or more of the following: severe swelling, sudden changes in the temperature of the feet, pinking or ecchymosis, pain and tenderness of the feet, and stiff toes sometimes with a toe drop. For lack of a better term we have called this condition chronic trench foot, as Theis[12] has described a chronic frostbite.

PROPHYLAXIS

Prophylaxis is important and methods for it have been published in Army journals.[13] The following considerations are of sufficient importance to warrant repetition:

1. The clothing and diet should be adequate.
2. Socks should be changed frequently.
3. Feet should be elevated as much as possible.
4. The feet should be exercised regularly. Greene reports on foot drill as employed in the trenches in 1918. During this exercise each man inspected his neighbor's feet and rubbed them in oil.
5. Socks and shoes should be removed at the first sign of swelling.
6. If trench foot is suspected there should be no rubbing with or without snow.
7. Sudden warming is dangerous.

SUMMARY AND CONCLUSIONS

The symptoms, clinical course, and possible etiologic factors in 125 cases of trench foot are presented. The patients were studied in a general hospital one to four weeks after the onset of their disease.

In the Italian Campaign this condition caused extensive disability. In those patients returned to duty, the average period of hospitalization was sixty days. Eighteen per cent required evacuation to the Zone of the Interior.

From observation of this group of patients, it appears that this is a condition caused primarily by ischemia due to capillary constriction resulting from long exposure to cold. This reaction is intensified by wet, stasis, and pressure. The continued ischemia produces varying degrees of capillary damage which upon the resumption of active circulation is manifested by edema and increased heat. In certain cases where edema persists, chronic inflammation with diffuse terminal fibrosis occurs and in more severe cases nonprogressive dry gangrene develops.

Simple conservative treatment with encouragement to walk at the earliest possible time seems to give the best results. Amputation of gangrenous parts was not necessary in any of these cases.

Prevention is probably not possible in all cases but education of the individual soldier in foot care, and the provision of loose, dry footgear should minimize the occurrence of this condition.

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OBSERVATIONS ON THE TREATMENT OF EMPYEMA THORACIS WITH PENICILLIN

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WHEN the sulfonamide drugs became available, the various derivatives were tried, both locally and systemically, in the treatment of empyema thoracis. Accumulated data on this problem prove that the almost universal use of the sulfonamide drugs for pneumonia has resulted in a marked decrease in the incidence of purulent pleurisy.[1-3] There is equally convincing evidence, however, that once pus has formed in the pleura neither the general nor the local administration of the sulfonamide drugs can be relied upon to effect a cure.[4] Toxic side actions preclude sufficiently large doses of the drug by systemic routes to influence materially an empyema after it has formed, and the local injections of the various sulfonamide drugs into the pleura have failed because their antibacterial action is inhibited when they are mixed with pus.

With the introduction of penicillin, interest has been revived in the nonsurgical treatment of empyema. Penicillin not only has a strong antibacterial action when given systemically, but also retains its potency in the presence of blood and pus. Moreover, this remarkable agent has a proved low toxicity for man and enormous doses can be employed, both parenterally and locally, without dangerous or distressing side effects. It appears, therefore, that penicillin is ideal for local injection into an empyema cavity, and when combined with systemic treatment a cure might be effected without surgical drainage when the infection in the pleura is caused by penicillin-vulnerable organisms. This optimistic outlook on the treatment of a hitherto surgical condition is substantiated by Tillett, Cambier, and McCormack,[5] who report eight patients with pneumonococcal empyemas treated by intrapleural injections of penicillin. In seven, the infection was eliminated by local therapy without surgical drainage; six recovered completely; and one patient with pyopneumothorax has a persistent pneumothorax, but the pyothorax has cleared satisfactorily. One patient, who in the opinion of these authors had insufficient initial treatment, suffered a relapse and required surgical drainage. Bennett and Parkes[6] report four cases. In one, a seropurulent, sterile effusion complicated a staphylococcal pneumonia. They believe this represented an early empyema aborted by sulfonamide therapy. A single injection of 20,000 units of calcium penicillin caused the

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local condition to disappear. Three other cases are described, two of which had collections of seropurulent fluid in the pleura containing beta hemolytic Streptococcus. Two local injections of penicillin caused the fluid to become sterile and cures seemed to be accomplished. The amount of penicillin given is not recorded. The fourth patient, a baby, had a massive Staphylococcus empyema. Systemic and local treatments with penicillin were employed and these authors state that after ten days of treatment the patient is convalescent and clinical and roentgen evidences "indicate that there will be no residual disease of lungs and pleura."

Undoubtedly as penicillin becomes generally available, more successful, nonsurgical cures of empyema will be described. This communication includes experiences with twenty-four cases of empyema. Thirteen patients received both systemic and local treatments; in five cases, intrapleural injections of penicillin were employed alone; and in six cases, only systemic administration of the drug was utilized. A much larger series of cases of empyema with chronic draining sinuses resulting from war wounds, which were operated upon with penicillin protection, are not included. The problems in this group are entirely different.

Important data are summarized in Table I.

It is evident that there is considerable variation in the amounts of penicillin given in each case and when intrapleural injections were employed there was no uniformity in the dosages or in the intervals between local administrations. The initial treatment in all but four cases was begun in other hospitals and the patients were referred to the Thoracic Surgery Section, Walter Reed General Hospital, after the acute phase of the disease had subsided. This accounts for the lack of a standard therapeutic plan.

The results from giving systemic injections of penicillin alone are summarized in Table II. Intramuscular injections were employed in all cases. In one case, Case 2, Table II, intravenous injections were also utilized.

SYSTEMIC ADMINISTRATION OF PENICILLIN

The results in only six cases are of little statistical value. The clinical impressions gained by studying this small series, however, are of some importance. One patient, case 3, Table II, would probably have died if large amounts of penicillin had not been given. It is interesting that only one positive culture was obtained from the pus in the empyema cavity after penicillin therapy was started. Local penicillin was not recommended because the fluid became sterile. The pus thickened rapidly, however, and required drainage. In this case, penicillin controlled a widespread Staphylococcus infection of the lungs. The empyema was relatively unimportant. In Cases 2, 4, and 5, Table II, the cultures of the pus were not altered but serious illnesses were converted into relatively innocuous ones by controlling the pulmonary lesion.

TABLE I

SUMMARY OF ENTIRE GROUP

CASE	PENICILLIN UNITS		OTHER MEDICATION	ORGANISM	RESULT	REMARKS
	LOCAL	GENERAL				
1	160,000	2,000,000	Serum Sulfadiazine	Pneumococcus type V	Thoracostomy; cure	Local penicillin daily for 4 days, 40,000 U. doses; operation 14 days after penicillin therapy started; pus became sterile but continued to form
2	460,000	None	Sulfadiazine	Hemolytic Str.	Chronic empyema required radical operation	Local penicillin given over period of 6 days, four doses of 100,000 U., followed by two injections of 30,000 U.; operation 17 days after penicillin therapy started; pus became sterile, lung would not re-expand, had broncho-pleural fistula
3	150,000	None	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	Local penicillin, initial dose 20,000 U., 7 days later, 75,000 U.; 7 days later 50,000 U.; operation 48 days after therapy started; pus temporarily sterilized but organisms re-appeared; original size of empyema cavity 2,000 c.c.
4	360,000	8,250,000	Sulfadiazine	Beta Str.; Staph. albus	Apparently cured without operation	Five intrapleural injections, two 100,000 P. doses and three 40,000 U. injections, over a period of 24 days; systemic penicillin therapy from March 1 to July 1, 1944; pus became sterile
5	100,000	None	Sulfadiazine	Beta hemolytic Str.	Thoracostomy; cure	Single dose of 100,000 U. of penicillin intrapleurally; operation 2 months after penicillin therapy; pus became sterile but drainage was necessary to evacuate it
6	240,000	400,000	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	Three doses of 70,000 U. and one injection of 50,000 U. of penicillin intrapleurally on 4 consecutive days; operation 4 days after penicillin therapy completed; pus became sterile and very thick
7	125,000	350,000	Sulfadiazine	Pneumococcus type V	Thoracostomy; cure	Local penicillin given on 3 consecutive days, operation 5 months later; after penicillin therapy, pus was sterile on culture most of the time; occasional positive cultures

8	80,000	4,160,000	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	40,000 U. penicillin, two doses in 3 days; operation 2½ months after penicillin therapy; pus sterile at intervals but thickened, and organisms re-appeared.
9	150,000	5,480,000	None	Beta Str.	Two operations to obliterate a chronic empyema cavity	Penicillin injected intrapleurally on 3 consecutive days; operation 4 months after penicillin therapy begun; pus in empyema cavity became sterile after the first injection of penicillin
10	600,000	930,000	Sulfadiazine	Anaerobic hemolytic Str.	Thoracostomy day of admission	Eight injections of intrapleural penicillin over period of 9 days; putrid pyopneumothorax, undrained for three weeks
11	30,000	2,840,000	Sulfadiazine	Hemolytic influenzae	Thoracostomy; cure	Organisms not vulnerable to penicillin; no effect on disease
12	110,000	685,000	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	Local penicillin on 4 consecutive days; operation 25 days after penicillin started; culture not effected by local penicillin
13	125,000	2,720,000	Sulfadiazine	Hemolytic Str.	Thoracostomy cavity not healed	Nine injections of local penicillin over a period of 21 days; operation 38 days after appearance of pleural fluid; after local penicillin most of cultures negative with organism visible on smear occasionally
14	100,000	770,000	Sulfadiazine	Hemolytic Str.	Apparently cured without operation	A single dose of 100,000 U. of penicillin intrapleurally; 3½ months' hospitalization; fluid remained clear, straw-colored and became sterile
15	150,000	None	Sulfadiazine	Staph. albus	Thoracostomy; cure	Local penicillin: initial injection of 50,000 U., 11 days later, 100,000 U.; operation 2 months after penicillin therapy started, pus became sterile
16	200,000	None	Sulfadiazine	Pneumococcus type V	Thoracostomy; cure	Local penicillin on 4 consecutive days; operation 1 month after penicillin started; pus became sterile but thickened and continued to form

TABLE I—CONT'D

CASE	PENICILLIN UNITS		OTHER MEDICATION	ORGANISM	RESULT	REMARKS
	LOCAL	GENERAL				
17	220,000	2,450,000	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	Local penicillin: 40,000 U. at 10-day intervals followed by 40,000 every other day for 3 days; operation 45 days after treatment begun; pus thick and sterile, had irrigations with saline
18	120,000	1,800,000	Sulfadiazine	Hemolytic Str.	Thoracostomy; cure	Three injections of 40,000 U. of penicillin intrapleurally on consecutive days; culture temporarily sterilized, operation 1 month after empyema found
19	None	670,000	Sulfadiazine	Unknown	Chronic sterile empyema; radical operation	Penicillin and sulfadiazine given before culture of fluid was established; sputum cultured hemolytic Str. 2 months from formation of fluid until operation
20	None	1,695,000	Sulfadiazine	Pneumococcus type I	Thoracostomy; cure	Empyema developed while patient was on penicillin therapy; total hospitalization 45 days; postoperative period 28 days
21	None	2,400,000	Sulfadiazine	Staph. aureus	Thoracostomy; cure	One positive culture showing Staph., all others negative; pus very thick, postoperative period 34 days
22	None	525,000	Sulfadiazine	Pneumococcus type XIX	Thoracostomy; cure	No effect on pus
23	None	720,000	Sulfadiazine	Hemolytic Staph. aureus	Thoracostomy; cure	No effect on cultures; massive empyema cavity, cavity healed in 60 days
24	None	640,000	Sulfadiazine	Nonhemolytic Str.	Apparently cured without operation	One culture of nonhemolytic Str.; all others sterile; fluid turbid but never thick

TABLE II

THE EFFECTS OF SYSTEMIC ADMINISTRATION OF PENICILLIN IN EMPYEMA

CASE	UNITS OF PENICILLIN	ORGANISM	COMMENT AND RESULT
1	670,000	Hemolytic Str. (?) in sputum	Penicillin and sulfadiazine given before an organism was found in pleural fluid; chronic sterile empyema requiring radical operation
2	1,695,000	Pneumococcus type I	Empyema developed while patient was receiving 25,000 U. of penicillin at 3-hour intervals
3	2,400,000	Staph. (?)	Thick pus found; one positive culture showing Staph.
4	525,000	Pneumococcus, type XIX	No effect on culture
5	720,000	Hemolytic Staph. aureus	No effect on cultures; massive empyema cavity
6	640,000	Nonhemolytic Str.	One culture of nonhemolytic Str., all others sterile; fluid turbid but never thick; apparently cured without drainage

The intramuscular administration of penicillin in large amounts will apparently inhibit the growth of bacteria in some infected effusions. This occurred in three of the six cases recorded. It is apparent, however, that this will not eliminate the necessity of removing frank pus from the pleura when it has formed. It is probable, however, that frequently pleural exudates which contain polymorphonuclear leucocytes and represent potential empyemas will remain sterile and be absorbed as a result of systemic penicillin therapy.

The results in patients treated with only intrapleural injections of penicillin are reviewed in Table III.

TABLE III

THE EFFECTS OF LOCAL PENICILLIN ON EMPYEMA

CASE	UNITS OF PENICILLIN	ORGANISM	COMMENT AND RESULT
1	460,000	Hemolytic Str.	Pus became sterile; chronic, sterile empyema requiring radical operation
2	150,000	Hemolytic Str.	Pus temporarily sterilized; cure after thoracostomy
3	100,000	Beta hemolytic Str.	Pus became sterile; drainage necessary because pus became very thick
4	150,000	Staph. albus	Pus became sterile on culture; operation necessary to evacuate thick pus
5	200,000	Pneumococcus type V	Pus became sterile; operation to remove thick sterile pus

INTRAPLEURAL PENICILLIN

Intrapleural injections of penicillin caused the disappearance of penicillin-vulnerable organisms from the pus in the empyema cavities of every patient so treated. Surgical drainage was performed in all of the cases in this group because the pus became so thick that removal by needle aspiration was difficult. Doubtless if more vigorous and frequent efforts had been made at needle aspiration, thoracostomy might have been avoided in some of these cases.

In two patients, the pleura was irrigated with saline solution to thin the exudate in an effort to make the aspirations more effectual. In both cases the pus was finally removed by surgical drainage.

All of the five patients who were treated with local penicillin alone had received sulfadiazine during the pneumonic stage of the disease. Intrapleural injections of penicillin had been started before the patient was referred to us. Since their general conditions were good and the pneumonias controlled, general administration of penicillin was omitted. It is our opinion, however, that the systemic administration of the drug should accompany local therapy.

COMBINED SYSTEMIC AND LOCAL PENICILLIN THERAPY

The results from the combination of systemic administration and intrapleural injections of penicillin are included in Table I, Cases, 1, 4, 6 to 14, 17, and 18. The obvious advantage of employing both routes is that the intramuscular or intravenous penicillin treatment is probably more effectual against the suppurative inflammation and pneumonic phases of the disease, whereas local injections of the drug may sterilize quickly the contents of the empyema cavity.

It is important to emphasize again, before discussing the effects of local and systemic penicillin on empyema thoracis, that it is possible that in some of our cases in which the pus became sterile, repeated thoracenteses may have eliminated the necessity for surgical drainage. The reasons for our reluctance to delay operation will become evident as the various cases are discussed.

In ten of thirteen patients treated with both intrapleural and systemic penicillin, the pleural fluids became sterile. In two patients, cases 7 and 8, Table I, organisms reappeared. In case 12, Table I, 110,000 units of penicillin, intrapleurally, on four consecutive days, did not cause hemolytic Streptococcus to disappear from the empyema cavity; 685,000 units of intramuscular penicillin were also given to this patient. The fluid in Case 11, Table I, was infected with *Hemophilus influenzae* and was not influenced by combined penicillin therapy. It is interesting that Tillett and his associates[5] describe a case in which thick pus, containing Pneumococcus, type XIX, and *Hemophilus influenzae* became sterile on culture after the introduction of intrapleural penicillin. Fleming's observations have indicated that *Hemophilus influenzae* is

not susceptible to the antibacterial action of penicillin. Tillett and co-workers[5] conclude that the high concentration of penicillin in the pleura or perhaps a strain of *Hemophilus influenzae* unusually susceptible to penicillin may account for their success.

Two penicillin cures are included in cases in which the patients received both intrapleural and general administration of the drug, and one patient developed chronic empyema while on combined therapy. These cases are presented later in the discussion.

The majority of the patients in this group had thick pus in the pleura when they were referred to Walter Reed General Hospital. Surgical drainage was performed to remove the pus without regard of its temporary sterility. In Cases 7, 8, 13, and 17, Table I, the period of hospitalization was prolonged unnecessarily because evacuation of the pus from the pleura was delayed, presumably because it was sterile.

COMMENT

Penicillin Cures.—Three patients escaped surgery. These cases deserve detailed consideration.

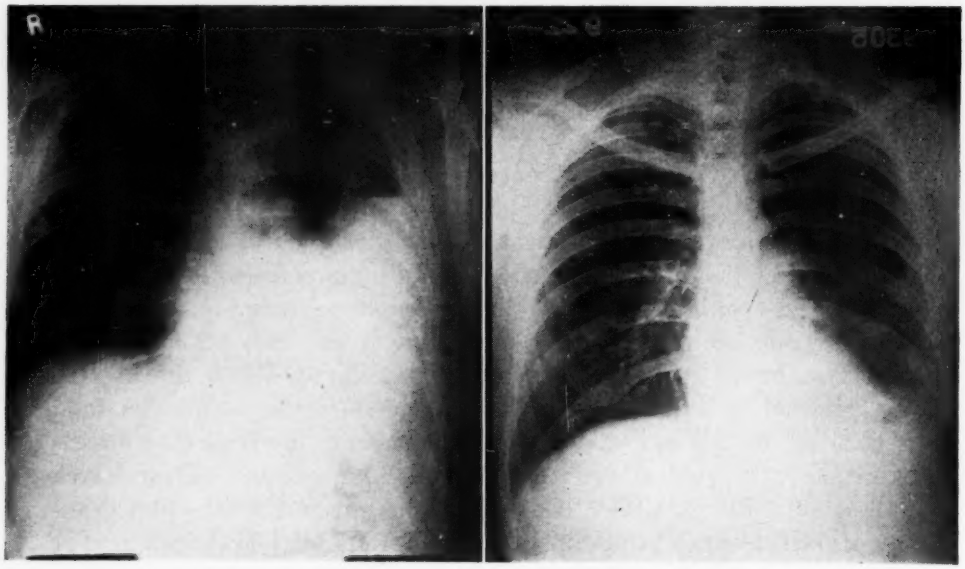
CASE 4 (Table I).—The patient developed a pleural effusion about one week after the onset of pneumonia. Cultures revealed *beta Streptococcus* and *Staphylococcus albus*. Five intrapleural injections of penicillin were given over a period of twenty-four days. Two initial doses of 100,000 units were followed by three injections of 40,000 units. The fluid removed by a thoracentesis is described as creamy brown. A total of 5,525 c.c. of this material was evacuated from the pleura by needle aspiration. The patient received intramuscular penicillin from March 1, 1944, until July 1, 1944. The pus became sterile after the first injection of intrapleural penicillin. A grand total of 330 intramuscular injections of penicillin (8,250,000 units) and eleven thoracenteses is a dear price to pay to avoid surgical drainage of an empyema cavity. Add to this six months of hospitalization and the nonsurgical cure loses its attractions (Fig. 1).

CASE 14 (Table I).—The second penicillin cure was accomplished in a patient who developed pneumonia which was first treated with sulfadiazine. On the fifth day of the illness, straw-colored fluid containing hemolytic Streptococci was aspirated from the chest. Systemic penicillin was begun and a single injection of 100,000 units of penicillin was given intrapleurally. The fluid remained clear and became sterile. Hospitalization was not prolonged and penicillin was discontinued as soon as the temperature, hemogram, etc. remained at normal limits for a week.

Critical analysis of this case indicates that this is not an example of an abscess being cured by penicillin, but rather it illustrates the value of local penicillin therapy in aborting an empyema. Pus was never present in the pleural cavity. Failure to make a clear and precise discrimination between an infected serous or serohemorrhagic fluid and a true, fully developed abscess of the pleura accounts for much of

the confusion in the past literature on the treatment of empyema. If this error is carried into the discussion of the value of penicillin in the treatment of empyema thoracis, observations on this phase of the subject will be of little value.

Case 24 (Table I).—The patient had pneumonia which was treated with sulfadiazine. After sulfonamide therapy the temperature subsided and improvement continued for ten days, followed by a recurrence of high fever. Fluid was found in the right pleura and 400 c.c. of cloudy yellow fluid were removed by thoracentesis. Cultures revealed a nonhemolytic Streptococcus. Penicillin by the intramuscular



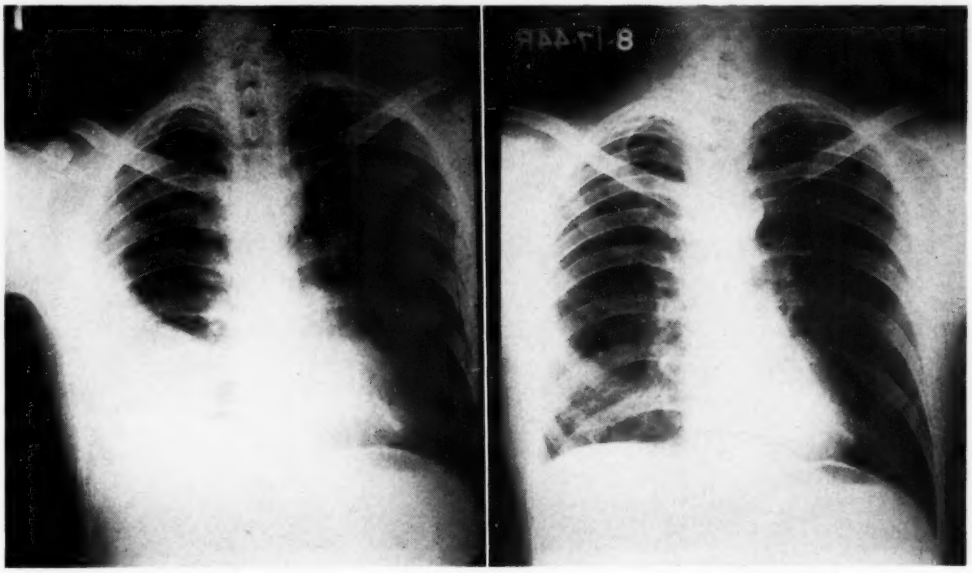
A. *B.*

Fig. 1.—*A*, Appearance of empyema after treatment by repeated aspirations and penicillin therapy was started. *B*, Roentgenogram four months later. The empyema seems to be cured by conservative management.

route was started the day before the fluid was discovered. A second thoracentesis, three days later, revealed sterile, turbid fluid which on smear contained 18,000 white blood cells with 100 per cent polymorphonuclear leucocytes. Intramuscular penicillin was continued for ten days. The fluid disappeared and the patient is fully recovered. No intrapleural penicillin was employed. This probably represents another example of the prevention of abscess formation in the pleura (Fig. 2).

Penicillin Sterile Chronic Empyemas.—There were three patients with chronic, sterile empyemas in the group of twenty-four cases. No new clinical spectacle was presented. They were the same gaunt, apathetic, hungerless individuals characteristic of old-fashioned, neglected empyemas of prepenicillin days. The chest wall is retracted, atrophy of disuse is evidenced in the puny muscles of the thorax, and the mechanical problem of cavity obliteration remains to be solved.

CASE 2 (Table I).—It is safe to conclude that penicillin therapy was extremely beneficial in Case 2. The patient developed an empyema complicated by a large bronchopleural fistula and was treated by closed drainage elsewhere before he was transferred to Walter Reed General Hospital. Three weeks after the original operation, wide dependent drainage was established. The fistula closed and the empyema cavity diminished in size. Healing progressed until the original 1,500 c.c. cavity measured 300 c.c. and then remained stationary. Radical surgical intervention was necessary to obliterate this cavity. The benefits of penicillin were confined to the control of a severe streptococcal pneumonia, which could easily have been fatal in the absence of penicillin therapy. There was probably no effect upon the complicating, purulent pleuritis.



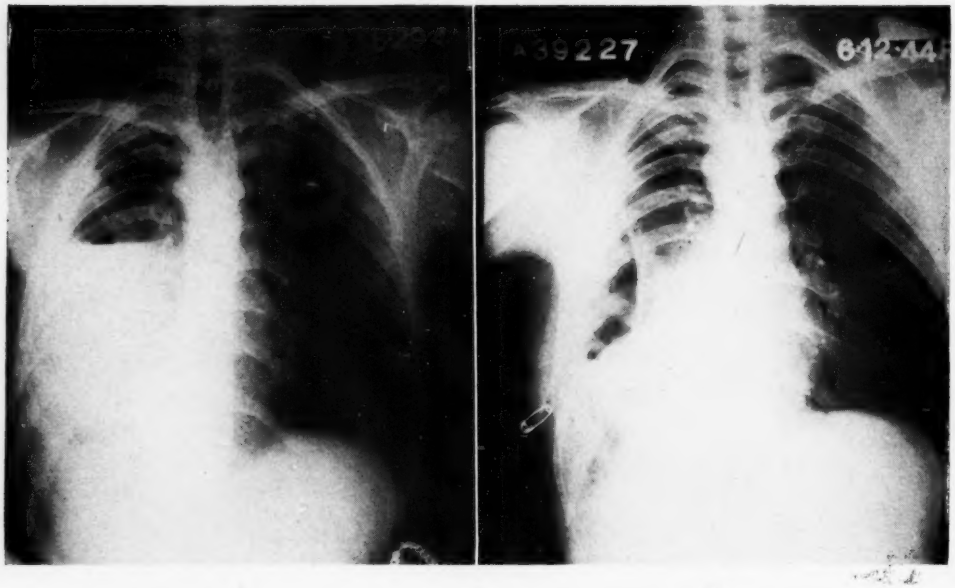
A. *B.*

Fig. 2.—*A*, Roentgenogram demonstrating a large collection of fluid in the right chest. The fluid was infected with nonhemolytic Streptococcus. *B*, Appearance of chest sixteen days later. Two thoracenteses and systemic penicillin therapy were employed. The cultures of the fluid became sterile without intrapleural injections of penicillin.

CASE 9 (Table I).—In Case 9, local and systemic penicillin was employed. The pus in the empyema cavity became sterile after the first intrapleural injection. The operation for the removal of the thick pus was delayed for four months, presumably because the cultures remained sterile. By this time the pleura was thickened and two operations were necessary to obliterate the cavity. Two thousand cubic centimeters of pus were removed at the first operation. Twelve thoracenteses had been performed during the trial of penicillin therapy. A total of 6,175 c.c. of fluid was aspirated from the chest during this same period (Fig. 3).

This is another example of carrying a good thing too far.

CASE 19 (Table I).—The patient became ill suddenly and had clinical and roentgen evidence of pneumonia. Fluid formed in the pleura, and systemic penicillin was employed before a culture of the fluid was obtained. The fluid remained sterile and consequently the true nature of the infection was never established. When the patient was referred to us there was a collection of thick, jellylike, sterile material in the pleura. It was impossible to remove it by needle aspiration. After careful studies to rule out the possibility of tuberculosis as an etiologic factor, the chest was opened and the organized, semiliquid mass was removed. The partially collapsed lung was splinted by a heavy layer of fibrous tissue, and it was necessary to perform a decortication operation to effect expansion of the lung.

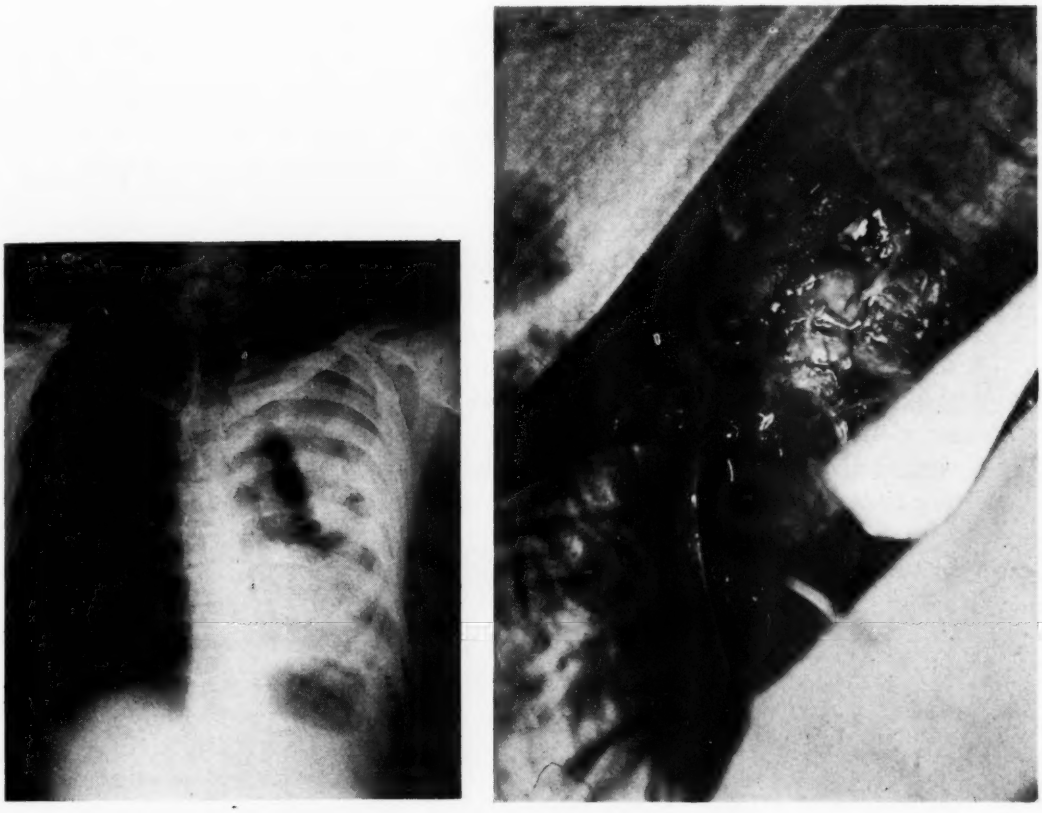


A. *B.*

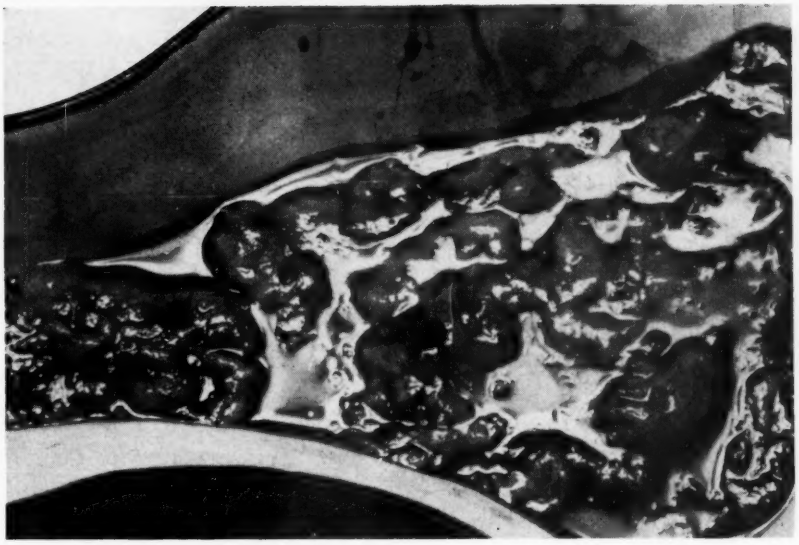
Fig. 3.—*A*, Roentgenogram of chest showing large empyema. Surgical drainage had been delayed four months because the pus remained sterile after local and systemic administration of penicillin. *B*, Appearance of the chest after surgical drainage to remove thick sterile pus; note thickened pleura. A second operation was necessary several months later to obliterate the cavity completely.

If vigorous attempts had been made to aspirate the sterile fluid from the pleura while it was still thin and the lung had re-expanded, a radical operation in this case would not have been necessary. The principal difficulty appears to be that a sterile empyema was erroneously considered as a cured empyema (Fig. 4).

Pyopneumothorax.—The pyopneumothorax treated by Tillett and coworkers,[5] in which the pyothorax was controlled but the pneumothorax persisted, has already been described. It seems unlikely that their patient is really cured. Fortunately, the infection responded to penicillin. In instances of putrid pyopneumothorax resulting from the rupture of a



A. *B.*



C.

Fig. 4.—*A*, The roentgen appearance of a chronic sterile empyema. *B*, Photograph of empyema cavity when it was opened at operation. *C*, Photograph of the puttylike sterile pus in the cavity.

lung abscess into the pleura, however, there is not only the problem of infection, but the immediate threat to life from altered intrapleural pressure.

Case 10 (Table I).—Case 10 furnishes an example of the overenthusiastic use of penicillin. The patient developed a pyopneumothorax. The pleura contained foul-smelling pus and cultures revealed anaerobic hemolytic Streptococci. A putrid infection of the pleura was treated by intramuscular penicillin, intrapleural penicillin, and sulfadiazine. Surgical drainage was delayed for three weeks (Fig. 5). Even in a mixed putrid infection of this type, penicillin may have some beneficial effects by altering or inhibiting some of the organisms growing in symbiosis with the common anaerobic bacteria usually responsible for necrotizing infections of the lungs. To delay drainage in this type of case, however, in the vain hope that penicillin or any other agent will effect a cure, indicates a complete lack of understanding of fundamental surgical principles.

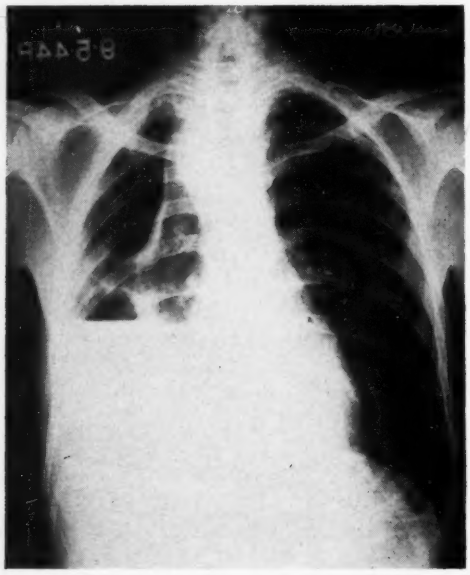


Fig. 5.—Roentgenogram of pyopneumothorax resulting from the rupture of a lung abscess into the pleura. The pus was putrid and contained anaerobic Streptococci. Drainage was delayed three weeks while local and systemic penicillin therapy was tried.

DISCUSSION

Before the importance of penicillin in the treatment of empyema can be evaluated intelligently, it is apparently necessary first to define empyema, second to enumerate the criteria for determining when an empyema is healed, and third to review the recognized fundamental principles of the treatment of empyema of prepenicillin times.

Definition of Empyema.—Empyema is defined by both Webster and Gould,[8, 9] as pus in a cavity, especially the pleural cavity. It is appar-

ent that serous or vaguely defined fluids should not be considered true abscesses of the pleura, and if cures are effected with penicillin they may represent aborted empyemas, but should not be considered as examples of nonsurgical cures of a mature abscess. It seems incredible that a sterile empyema could be considered a cured empyema, yet disastrous procrastination in cases in which the pleura was filled with sterile pus has occurred in several of our cases.

Graham[10] has repeatedly emphasized the importance of distinguishing between infected effusions and fully developed abscesses of the pleura, in fact, the successful management of suppurative pleurisy depends upon this distinction. Premature open drainage of an infected effusion during the formative stage of an empyema exposes the patient to the dangers of interference with normal intrathoracic pressure relationships. Conversely, evacuation of thick pus must be performed early enough to prevent chronicity. Even when the pus of a pyogenic infection has been temporarily sterilized by chemotherapy, the problem of obliteration of an abscess cavity will remain to be solved. If this fundamental principle is accepted, it is essential to establish criteria to determine when an empyema cavity is really healed.

When Is an Empyema Healed? An empyema cavity cannot be considered cured until the cavity itself is obliterated. There is only one thoroughly reliable method to determine this, namely, by actual measurements of the cavity itself. Roentgenograms of the chest are helpful but experienced radiologists are aware that fairly large amounts of fluid may not be visible on ordinary roentgenographic examinations. For example, Rigos[11] has pointed out that in roentgenograms taken in the upright position, effusions of less than 400 c.c. are often difficult to detect. It is apparent, therefore, that improvement in the roentgenographic appearance of the chest following penicillin therapy for empyema does not necessarily constitute positive proof of a cure. Experienced surgeons are fully aware of the possibilities of recurrence of an empyema months or even years after the original illness. The term "cure" should be used with considerable caution, particularly when so-called nonsurgical measures of local injections and aspiration of the empyema are employed. In these cases accurate measurements of the cavity are impossible to obtain, and clinical and roentgenographic evidence may be temporarily misleading.

Fundamental Principles of Treatment of Empyema Thoracis.—During World War I, in connection with the activities of the Empyema Commission, Graham and Bell[12] outlined the principles for the treatment of empyema. The introduction of the sulfonamide derivatives has in no way altered their conclusions. It is important to re-examine these principles and to determine whether or not penicillin therapy will effect them. The two most important principles are, first, not to create an open pneumothorax during the time pneumonia is present; and second,

to provide adequate drainage when a true abscess of the pleura has formed. A third principle is to maintain the nutrition of the patient.

The fundamental surgical physiology of the chest is now so generally appreciated that the injudicious use of open drainage in cases of empyema before a frank abscess is formed is rarely encountered. It is not necessary, therefore, to dwell upon the dangers of open pneumothorax during the formative stage of an empyema.

The second principle, namely, the maintenance of adequate drainage when drainage becomes necessary does, however, require comment in connection with the use of penicillin. The primary object of all endeavors in the treatment of empyema is, of course, to save life. The second most important goal is the prevention of chronic empyema. The great majority of cases of chronic empyema have resulted because the drainage has been inadequate.

The third principle, namely, maintenance of nutrition, requires no comment in this vitamin-protein-calorie conscious era.

Adequate Drainage.—Obviously, any type of drainage which results in a cure is adequate, whether it is by needle aspiration, closed drainage, or rib resection. Controversial opinions concerning which method of drainage should be employed have occupied a prominent place in literature on empyema. It appears that the greatest difficulty has been that the exponent of a particular technique insists upon applying his method to every case of empyema. This attitude is as unreasonable as attempting to treat all fractures with traction alone, exclusively by plaster casing fixation, or by any other single method.

Instances of successful cures by thoracentesis were recorded long before either the sulfonamides or penicillin were available, notably in cases of pneumococcal empyema where successful aspiration treatment may be expected in from 10 to 25 per cent of cases.[13] This is particularly true in children. It is probably safe to conclude that with the early use of intrapleural penicillin injections, healing will occur in a fair percentage of cases after a few aspirations. The attractive possibility that intrapleural injections will eliminate the necessity for surgical drainage in many patients has, however, in our series of cases, resulted in the condition becoming chronic in two cases and too long periods of hospitalization in others. When surgical drainage is performed there is reasonable assurance of healing in a period of five or six weeks. Naturally the question of relative risk to life in the two plans of treatment must be taken into account. The judicious use of needle aspiration carries practically no operative risk. If, however, fanatical attempts, as for example eleven thoracenteses and 330 injections of penicillin intramuscularly, are carried out to effect a nonsurgical cure, definite dangers are introduced, principally, the excellent chance of the condition becoming chronic. There is also the inconvenience and discomfort of repeated thoracenteses and in this con-

nection it is worth recording that in two of our patients to whom frequent intrapleural injections of penicillin had been given, the drug was injected into the lungs by mistake. These accidents were accompanied by rather sharp, disagreeable, but not severe, reactions.

It is fair to conclude that a properly performed intercostal drainage or even rib resection is no more painful than a needle aspiration of the chest. There is, however, more postoperative pain and discomfort from tube drainage than from aspiration, but this is usually not prolonged and is controlled easily. The fear of the needle, which most patients develop after a long course of penicillin treatment and repeated thoracenteses, presents a much more serious clinical problem than the control of postoperative pain.

Irrigation of Empyema Cavities.—The general use of Dakin's solution for an irrigating agent during World War I was considered a dramatic advance in the postoperative management of empyemas. The principal advantage of this solution is not so much its antibacterial action but its ability to loosen and dissolve the necrotic tissue after drainage has been accomplished. In this connection, it is worth recording that properly prepared Dakin's solution can still be considered a highly satisfactory agent for this purpose and, if employed vigorously enough, will sometimes cause temporary sterilization of cultures obtained from drained empyema cavities. In fact, Keller[14] maintained Dakinization of chronic empyema cavities until three consecutive negative cultures were obtained before proceeding with a radical operation to obliterate the space. Azochloramide is also an excellent solution for postoperative irrigations. It is evident from our findings and from the reports of others that the instillation of penicillin into an unopened empyema cavity will practically always cause temporary sterilization of its contents, provided the infection has been caused by a penicillin-vulnerable organism. It is equally evident, however, that even a sterile empyema may be made up of thick, puttylike pus and an incidence of three chronic empyemas in a series of twenty-four cases illustrates dramatically the danger of delaying evacuation of the pleura too long.

SUMMARY

In considering the possibilities of altering the management of empyema by the use of penicillin, the questions of adequate drainage and irrigation are most important. The prevailing tendency in penicillin-treated empyemas has been to delay drainage and therefore decrease the chance of premature operations in cases where the abscess is in a formative stage. Moreover, the use of penicillin will control quickly the pneumonic stage of the disease and consequently decrease the risk of any type of surgical intervention.

There is indisputable evidence that penicillin is a valuable adjunct in the treatment of empyema thoracis caused by penicillin-vulnerable organisms. Systemic administration of the drug alone will sometimes

cause cultures of the fluid in the chest to become sterile. Intrapleural injections of penicillin will temporarily sterilize an empyema cavity in the great majority of cases. It is our impression, however, that combined systemic and intrapleural administration is desirable because the combined routes effect both the pulmonary and pleural lesions.

Probably penicillin will be extremely effectual in aborting empyema formation, but once pus is formed, and continues to form, even if sterile, the problem of cavity closure cannot be ignored. If healing is not rapid with disappearance of pus when conservative measures are employed, it is our opinion that surgical drainage is mandatory. Failure to observe the fundamental principle of adequate drainage of any pyogenic empyema, even if the pus is rendered temporarily sterile by penicillin, will invite the serious complication of chronicity. This occurred in two of twenty-four cases which we have observed. It should again be emphasized that the introduction of penicillin has not altered in any way the fundamental principles of treatment of empyema; only the details are changed.

As experiences with penicillin therapy accumulate, the therapeutic approaches will become more standardized. Based upon our own observations, a tentative working plan is suggested:

1. That an injection of intrapleural penicillin be given as soon as infected fluid appears in the pleura. It is important that local treatment be withheld until the organisms in the fluid are identified. This precaution will prevent waste of penicillin in infections which do not respond to the drug. An initial positive bacteriologic diagnosis will also rule out the possibility of a tuberculous effusion or empyema which may deserve consideration if no other bacteria are found.

2. If systemic penicillin has not been employed during the pneumonic stage of the disease, it should be combined with the local therapy. It might be argued that by the time an empyema becomes evident the pneumonic phase of the disease will have subsided and, therefore, systemic administration of penicillin would be superfluous. Florey and Cairns[16] have emphasized the advantages of combined administration of penicillin in severe war wounds. The chief advantage of the systemic route is that the blood will contain a bacterial inhibitory substance which may control a spreading cellulitis or invasive infection. It is our opinion that the same principle is sound in the management of pneumonia complicated by empyema. This is particularly true in cases of streptococcal or staphylococcal empyema.

3. Probably three injections of 50,000 units of penicillin on alternate days are sufficient for local treatment. Before the penicillin is injected into the pleura, as much pus or infected fluid as possible should be removed by thoracentesis.

4. If pus continues to form and thicken, surgical drainage should be established. A *sterile* empyema is not a *cured* empyema, and evacuation

of frank pus should not be unduly delayed because organisms cannot be found after the penicillin treatment has been started. Our experiences confirm the statement included in the report of the Committee on Chemotherapeutic and Other Agents, Division of Medical Sciences, National Research Council, by Keefer and associates,[15] namely, that even when the pus in an empyema cavity is sterilized by penicillin it often becomes so thick that thoracostomy is necessary to furnish adequate drainage.

As an adjuvant to skillful surgery, penicillin has already revolutionized the management of many surgical diseases of the thorax. In their excellent paper on *Major Complications of Penetrating Wounds of the Chest*, D'Abreu, Litchfield, and Hodson[17] give a brilliant account of the value of the mold both as a prophylactic and as a therapeutic agent. There is every reason to predict that with "penicillin protection" the dangers of practically all operations upon the chest will be reduced. This communication is not intended to depreciate the value of this remarkable agent, but to emphasize that it will not afford protection from violations of the fundamental principles of surgery.

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COMPLICATING FACTORS IN THE SURGICAL MANAGEMENT OF VARICOSE VEINS

With Special Reference to Interruption of Sympathetic Nerve Impulses as an Adjunct in Treatment

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SUCCESSFUL results in the treatment of varicose veins of the legs depend upon complete obliteration of the principal components of the superficial venous circulation, the great saphenous system. Elimination of such an extensive venous network requires radical measures and it is to be emphasized that best results can be obtained only by a radical surgical technique.

Although numerous technical procedures have been developed through the years, only two have survived critical evaluation to the present time. The first is high ligation and division of the great saphenous vein combined with retrograde chemical sclerosis of the entire vein and its main tributaries. The operative technique of this procedure has been described in adequate detail[1-9] and needs no further elaboration except for one technical feature which deserves special mention. Sclerosis of the saphenous vein and its tributaries must be complete. Accomplishment of this important detail can be assured by employing the segmental sclerosis method of Pratt.[1] By this procedure, thorough sclerosis of the entire saphenous vein, from ankle to groin, can be obtained by passing a ureteral catheter a distance of 40 to 60 cm. down the distal venous segment after division of the main trunk at the fossa ovalis. The sclerosing agent is injected continuously as the catheter is withdrawn slowly upward, thus inducing thrombosis throughout the entire venous trunk. The advantage of catheterization is obvious: the common sclerosing materials act quickly and become rapidly fixed at their point of contact with the intima, so that retrograde injection at the fossa ovalis by needle or cannula results in obliteration of only a short segment of vein in the thigh, incomplete effect being exerted below the knee. Catheterization need not be confined to the main saphenous vein but may be applied to large tributaries as well. Such a maneuver involves the use of large quantities of sclerosant, as much as 30 c.c. of sodium morrhuate solution being injected into a single extremity.

The second method is the Mayo technique, consisting of the actual removal of long venous segments after stripping the varicosities free from their subcutaneous attachments. While not widely employed in

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recent years, it has been carefully evaluated lately by Grimson[10] in a large series of patients with gratifying results.

A high percentage of satisfactory results can be achieved by the combined ligation-division and massive sclerosis method of treatment. Certain complicating factors may be encountered, however, which interfere with the performance of an ordinarily simple operation and which may prolong or harass the convalescence. It is these complications in the treatment of varicose veins with which the ensuing discussion is concerned.

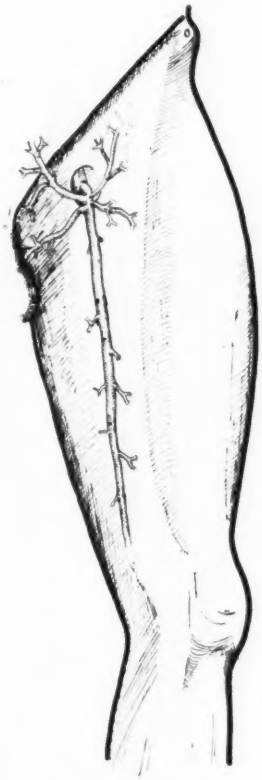

Fig. 1.—The usual anatomic pattern of four saphenous tributaries at the fossa ovalis.

ANATOMIC VARIATIONS

Anomalies of venous channels are common. From the standpoint of the great saphenous vein, three varieties in particular deserve mention: First the irregularity of relationships of the tributary channels at the fossa ovalis, second the occasional occurrence of an abnormally low saphenofemoral junction, and third the presence of tortuosity of the vessels.

Tributaries.—Normally, the main saphenous vein receives four well-defined venous trunks at, or very near, its entrance into the deep femoral vein (Fig. 1). These branches in most instances join the saphenous superficial, or external, to the fossa ovalis and, hence, super-

ficial, to the fascia lata. Ligation, division, catheterization, and retrograde sclerosis of each individual tributary, as well as the main saphenous vein itself, are important technical steps in the prevention of recurrences and can be done easily as long as the normal anatomic pattern prevails. Occasionally, however, anomalies of location place certain contributary trunks deep within the fossa ovalis itself. Such an arrangement complicates the operation by making the abnormal branches difficult to visualize to the extent that they may be entirely overlooked in the dissection. Case 1 illustrates this problem.

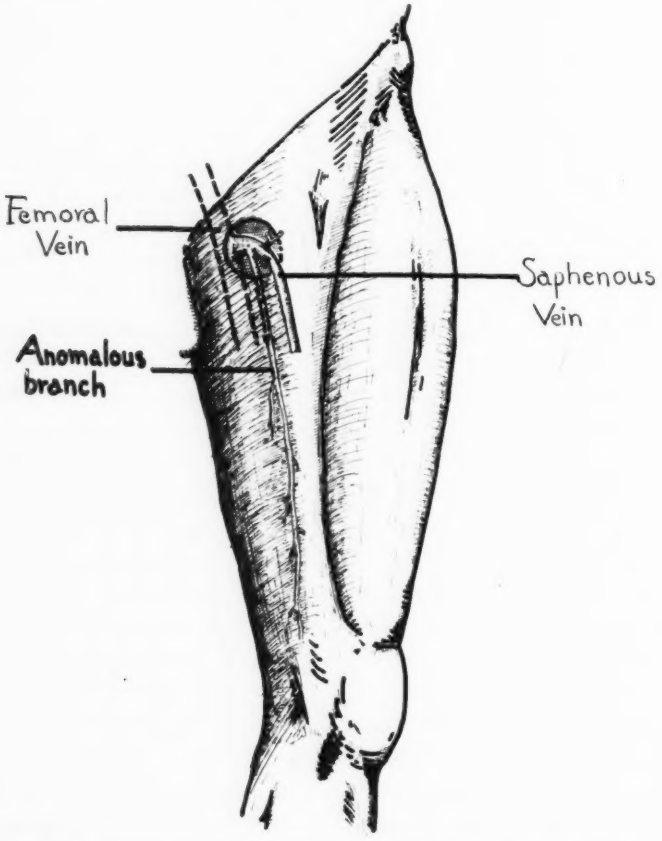

Fig. 2.—Anomalous anatomic arrangement showing one large tributary entering saphenous vein deep to fascia lata (see Case 1).

CASE REPORTS

CASE 1 (Roper Hospital No. 23262).—A 43-year-old Negro woman entered the hospital in October, 1943, presenting an indolent, fibrotic ulcer on the medial surface of the lower third of her right leg. Varicose veins were well defined and the history revealed that inadequate treatment had been attempted some eight years previously. After routine preoperative study by tourniquet tests, ligation-division and catheter injection of the saphenous vein were done at the fossa ovalis and just above the knee. During the dissection in the region of the saphenofemoral junction, it was noted that only two tributaries were demonstrable. After division of the superior margin of the fascial ring constituting the fossa ovalis, an

abnormally situated venous trunk was seen to enter the saphenous vein almost flush with the saphenofemoral torcula (Fig. 2). This was divided close to its point of entrance into the saphenous vein and its distal segment was catheterized in the usual manner. Palpation failed to detect the presence of the catheter in the upper 15 cm. of the thigh, but below this level the catheter could be palpated lying immediately beneath the skin. Such a situation indicated that the anomalous branch, although a part of the varicose saphenous system, was located deep to the fascia lata in the upper portion of the thigh, as illustrated in Fig. 2. Ligation, division, and retrograde sclerosis of the anomalous vessel and the other principal superficial trunks resulted in prompt healing of the ulcer.

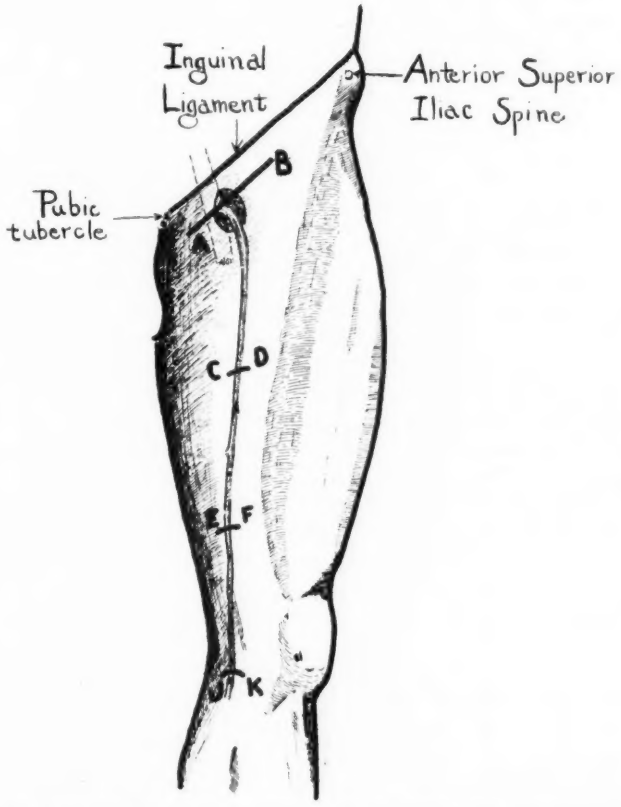

Fig. 3.—*AB* represents routine transverse incision for exposure of saphenofemoral junction. *CD*, *EF*, and *JK* represent sites of election for re-exposure of saphenous vein when necessitated by obstruction to catheterization (see Case 3).

Low Saphenofemoral Junction.—In the great majority of patients, the main saphenous vein enters the femoral vein, via the fossa ovalis, about one inch below the inguinal ligament. As illustrated by Case 2, an abnormally low junction sometimes occurs. This variation can be annoying to the operator who prefers a transverse incision (Fig. 3), but of less significance when a vertical incision is made (Fig. 4); in either case, the existence of such an anomaly is confusing.

CASE 2 (Roper Hospital No. 20175).—A 26-year-old white medical student was admitted in June, 1943, because of massive saphenous varicosities of both legs, without pigmentation or ulceration. After preliminary tourniquet studies, each

leg was operated upon by the ligation-division and injection technique. Because of the tremendous degree of involvement, two procedures were carried out three days apart. A transverse incision was made in the left thigh one inch below and parallel to the medial one-half of Poupart's ligament. The fascia lata was exposed throughout the entire field, but the saphenous vein could not be located after careful search. Thereupon the fascia lata was divided vertically and the femoral vein was brought into view. By following this vessel distally the saphenous vein was found to enter at a point three and one-half inches below the level of the incision, strong downward retraction of the lower edge of the wound being necessary to expose the junction. The operation was then completed in the routine manner. An excellent result was obtained in each leg and the patient subsequently passed a military physical examination with ease.

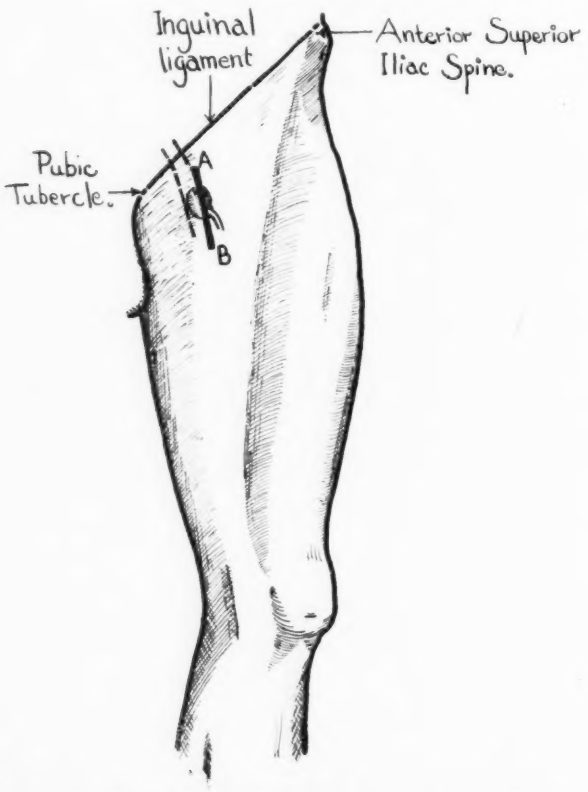

Fig. 4.—Vertical incision for combined high saphenous ligation-injection and femoral periarterial sympathectomy (see Case 7).

Extreme Tortuosity.—A complicating factor of considerable magnitude is the presence of marked tortuosity and multiple sacculations of the main saphenous vein. As previously stated, complete obliteration of the principal components of the saphenous system is the only guarantee of cure of varicose veins. If this is to be accomplished by injection of sclerosing material, virtually the entire intimal lining of the saphenous trunk and its largest tributaries must be subjected to contact with the thrombosing substance employed. Such an accomplishment is rendered difficult by the presence of tortuosity. When injec-

tion by needle or cannula is attempted, the greater intimal surface created by the existence of numerous saccular dilatations causes pooling and fixation of the sclerosing solution in a short high segment of vein and prevents dissemination distally from the point of injection. When catheterization is employed for the purpose of segmental sclerosis, arrest of the catheter usually occurs in the tortuous channel and its downward progress is halted. This difficulty frequently can be overcome by reversing the usual technique and re-exposing the saphenous trunk in the calf or at the knee (Fig. 3). Having done so, the venous channel is catheterized from below upward and the catheter usually can be made to appear at the point of saphenous division in the groin incision. Segmental sclerosis is then accomplished by slow, steady injection as the catheter is withdrawn gradually downward along the saphenous vein. Any remaining uninjected segments of the main venous trunk are treated in a similar manner until complete sclerosis has been obtained.

CASE 3 (Roper Hospital No. 19046).—A 45-year-old white woman was admitted May 31, 1943, for treatment of extensive bilateral varicose veins. After the usual preoperative evaluation of her deep venous circulation and arterial status, each leg was treated by the combined ligation-division and injection technique at separate operations five days apart. The procedure was uneventful in the case of the right leg, but tortuosity and sacculation of the left main saphenous were pronounced and caused arrest of the catheter 5 cm. from the fossa ovalis. To quote from the operative record, "so the latter (great saphenous vein) was re-exposed by a transverse incision just above the medial condyle of the knee (Fig. 3). The catheter was then passed upward from the knee and the entire segment of saphenous from femoral junction to knee was injected with 10 c.c. of sodium morrhuate." The remaining varicosities below the knee were then injected by catheterization to the ankle. The patient recovered uneventfully and is now judged to be cured.

COMPLICATIONS AMENABLE TO TREATMENT BY INTERRUPTION OF SYMPATHETIC NERVE IMPULSES

In recent years, the role of the sympathetic nervous system in disorders of the peripheral circulatory apparatus has received increasing attention. Regarding certain complications of varicose veins, sympathetic nervous activity is intimately concerned as a part of the pathologic process in three specific instances: (1) vasoconstriction, (2) pain of vascular origin, and (3) local hypoxia. Each of the following complications is characterized by one or more of these three autonomic nervous factors and each can be improved or completely abolished by interruption of the regional sympathetic impulses.

Thrombophlebitis.—In 1940, Ochsner and DeBakey[11] emphasized the strong element of vasospasm in the presence of acute inflammatory obstruction of the deep veins of the lower extremity. It is now generally accepted that the presence of inflammatory thrombosis, or even perivascular inflammatory reaction without actual venous obstruction, in-

itiates reflex vasoconstrictor impulses which are mediated by the sympathetic nervous system. Reflex impulses of autonomic origin tend to be discharged *en masse*. As a result, the vasoconstriction initiated by a femoral thrombophlebitis is widespread in the affected extremity and involves veins as well as arteries. Abolition of the vasospasm results promptly when novocaine infiltration of the lumbar ganglionated sympathetic chain is performed.

Spontaneous idiopathic thrombophlebitis is common in varicose veins, usually involving the main saphenous in its entirety or in either the thigh or leg segments. Accompanying signs of inflammation are prominent, consisting of redness along the distribution of the vein, increased local heat, and a palpable swelling of the involved segment. Pain is variable but is generally present and sometimes may be severe. Edema of the extremity may be a feature. In the presence of such a process, the methods of treatment of the varicosities vary from the usual routine procedure. In so far as it is possible, treatment should be directed toward obliteration of the saphenous tree as well as toward the thrombophlebitis. The latter condition presents two problems—prevention of embolism and control of the painfully disabling inflammatory process. Both can be accomplished by excision of a two-inch segment of the main saphenous vein at its junction with the femoral vein and ligation of the divided ends. Rather dramatic relief of pain and rapid subsidence of the inflammatory process occur following division of the venous trunk, a fact substantiated by Welch and Faxon,[12] Fine, Frank, and Starr,[13] and Heyerdale and his co-workers.[14] The mechanism of pain relief is thought to be due to interruption of the sympathetic fibers which pass alongside of the vein, lying in its tunica adventitia. This explanation is based upon the frequently observed abolition of pain in deep femoral thrombophlebitis after procaine block of the lumbar sympathetic ganglia. Actually, sympathetic ganglion block is a rational form of treatment of saphenous thrombophlebitis inasmuch as it accomplishes both relief of pain and subsidence of the inflammatory process. It does not, however, offer full protection against proximal propagation of the thrombus and possible embolism. For this reason, excision and ligation constitute a preferable method of treatment when the deep circulation has been found to be patent by clinical and venographic studies.

When excision-ligation has been chosen as the method of treatment of acute saphenous thrombophlebitis, the question arises as to the need for simultaneous retrograde sclerosing injection. In the presence of involvement of the entire great saphenous trunk by the phlebitis, additional inflammatory activity of a chemical sort should be avoided. As the original phlebitis subsides following ligation and division, an extensive degree of obliteration of the venous channel results from fibrosis. When, however, only a portion of the vein is affected by the phlebitis, sclerosing injection of the uninvolved segment should be done at the

time of ligation and division. More than one incision may be necessary to isolate the uninvolved segment for catheterization, as occurs when only that portion of vein from the saphenofemoral junction to the knee is the site of an inflammatory obstruction. Cases 4 and 5 illustrate two variations encountered in managing superficial thrombophlebitis.

CASE 4 (Roper Hospital No. 24127).—A 44-year-old Negro woman entered the hospital Nov. 17, 1943, complaining of tenderness, pain, and swelling of the left leg. One week previously the patient suffered from chills and fever, during which time a small ulcerative lesion appeared above the lateral malleolus of the left ankle after a slight injury to that region. On examination, the entire great saphenous vein was found to be indurated, hot, and tender to manipulation. Inguinal lymph node enlargement was present. The patient was afebrile. Division of the saphenous trunk was done at the fossa ovalis and ligation of the divided ends was accomplished without retrograde injection. Twenty-four hours after operation there was complete disappearance of both subjective and objective discomfort and the phlebitis had subsided satisfactorily. The patient was discharged forty-eight hours after operation with complete subsidence of the phlebitis and entirely free of pain. Follow-up observations in the outpatient department found her free of demonstrable varicosities. The small ulcer noted on admission healed within a week.

CASE 5 (Roper Hospital No. 23983).—A 59-year-old white woman was admitted Nov. 15, 1943, because of a diffuse cellulitis of the lower half of her left leg, extending over the proximal portion of the dorsum of the foot. There was present, also, a well-defined acute thrombophlebitis of the great saphenous vein from ankle to knee. The vein was hot and extremely tender and was palpable as a thickened indurated cord. There was no involvement within the thigh. The patient had no history indicating the source of her infection and no portal of entry was discovered on physical examination. In view of the rather virulent nature of the cellulitis, it was decided to defer ligation-division of the saphenous vein. Accordingly, novocaine infiltration of the left lumbar sympathetic ganglia was done shortly after admission. One hour later she stated that she was "considerably relieved" of pain. Seven hours later there was evidence that the cellulitis was subsiding and at the end of twenty-four hours it had been reduced to 50 per cent of its original extent. The sympathetic block was repeated after thirty-six hours and was followed by complete disappearance of the cellulitis. Three days after admission, the saphenous vein was barely palpable below the knee, practically all induration having subsided. At this time operative treatment was carried out as follows: the main saphenous trunk was exposed just above the knee and ligation-division completed, thus isolating the lower segment previously involved by the phlebitis; retrograde injection was not done. Exposure of the saphenofemoral junction was then accomplished and ligation-division again carried out. In this manner, the thigh segment of vein, which had not been affected by the phlebitis, was isolated and subjected to catheterization and segmental injection of sclerosing material. Convalescence was uneventful and the patient is now considered to be free of demonstrable varicose veins.

Lymphangiitis and Cellulitis.—Diffuse reticular lymphangiitis, manifested by marked hyperemia, brawny induration, increased local heat, tenderness, and pitting edema of the involved area, is seen rather often in the lower half of the leg in the presence of venous stasis. Frequently no bacterial portal of entry can be demonstrated or a small ulcer may be an associated finding. Acute superficial thrombophlebitis

sometimes precedes and accompanies the process. In either case, procaine infiltration of the ipsolateral lumbar sympathetic ganglia is of great benefit and is followed by rapid resolution of the inflammatory process. Case 5 is a typical example of favorable response to sympathetic block. The following case represents a diffuse inflammatory process of different etiology, successfully treated by paravertebral novocaine infiltration.

CASE 6 (Roper Hospital No. 17575).—A 58-year-old white man was admitted Feb. 7, 1944, because of bilateral varicose veins. A scaly varicose eczema was present over the right ankle and lower leg and showed several weeping areas of superficial denudation. After three days of preliminary treatment of the eczema by warm moist dressings, bilateral saphenous ligation and retrograde injection were performed; catheters were passed 60 cm. down each vein and 10 c.c. of 5 per cent sodium morrhuate solution were injected segmentally into each vein. The resulting chemical thrombophlebitis was marked and considered satisfactory. On the second postoperative day the patient presented evidence of an extensive spreading cellulitis from ankle to knee of the right leg and complained of much pain in the area. The temperature was elevated to 102° F. A right paravertebral novocaine infiltration was done at the levels of the first through the fourth lumbar vertebrae. Within twenty-four hours, the entire inflammatory reaction had subsided and the patient was free of discomfort. Follow-up studies in the outpatient department showed healing of the dermatitis to be slow but finally complete. The varicose veins were considered cured.

Varicose Ulcer.—The underlying factor in the causation and perpetuation of varicose ulcer is chronic tissue hypoxia incident to venous stasis. When of long duration, hypoxia results in tissue fibrosis. The presence of fibrosis about an ulcer renders the latter extremely resistant to cure, even after successful elimination of venous stasis, because of the pronounced diminution in vascularity of the fibrous tissue. This factor can be combated to a large extent by interruption of the sympathetic impulses of the affected extremity.

In cases of long-standing, indolent, varicose ulcer, combined sympathectomy and saphenous ligation-division are recommended. The rationale of combining these procedures is based upon two factors resulting from the vasodilatation produced by sympathectomy: (1) tissue oxygen tension is rapidly increased and sustained, and (2) increase in phagocytosis and collateral circulation is accomplished. The first factor is self-evident while the second has been demonstrated in experimental observations of "local shock" by Thomson, Helwig, and Sire.[15] The increase in vascularity of the extremity, when combined with obliteration of the varicose veins, aids materially in healing the indolent types of ulcer.

Technically, sympathectomy can be performed in one of two ways in the lower extremity. Either lumbar ganglionectomy or femoral periarterial sympathectomy will eliminate impulses of sympathetic nervous origin in the legs. A third method, crushing the mixed peripheral nerves, is hardly applicable to leg ulcers because of the exten-

sive loss of motor function incurred by the operation. Periarterial sympathectomy produces only temporary vasodilatation, regeneration occurring within a few weeks. However, its effects seem to be of sufficient duration to augment the elimination of tissue hypoxia and, when combined with the operative cure of varicose veins, to permit healing of the ulcer to begin. Once venous stasis has been abolished the healing process progresses usually to complete cure. In the event of recurrence of the ulcer, the more formidable operation of lumbar ganglionectomy deserves consideration.

CASE 7 (Roper Hospital No. 22886).—A 41-year-old white man entered the hospital Oct. 9, 1943, because of left saphenous varicosities and severe varicose eczema. The lower half of the left leg was red, indurated, and leathery and showed a superimposed scaling ulcerative dermatitis. Marked fibrosis was present and, with the patient standing, a suffused cyanotic appearance of the leg developed, indicating advanced venous stasis and tissue hypoxia. After twelve days of preliminary treatment of the dermatitis, combined femoral periarterial sympathectomy and saphenous ligation-division and injection were performed through a vertical incision (Fig. 4) in Scarpa's triangle. A ureteral catheter was passed distally 20 cm. from the fossa ovalis, and 10 c.c. of 5 per cent sodium morrhuate solution were injected as the catheter was withdrawn upward. The main saphenous vein was exposed again just above the medial condyle of the femur and catheterization-injection distally was repeated. The patient developed a very satisfactory chemical thrombophlebitis and the skin temperature of the toes became greater than that of the unaffected extremity. Prompt healing of the ulcerations and dermatitis and disappearance of the varicose veins resulted.

Severe Postoperative Vasospasm.—Generalized spasm of the arterioles and small veins of an extremity is initiated by an inflammatory process within one of the main venous trunks. Experimentally, DeBakey, Burch, and Ochsner[16] have shown that a chemical endophlebitis, localized to a given segment of a large vein, causes vasospasm of sufficient severity to reduce greatly the volume of peripheral pulsations in the affected limb. They have demonstrated also that the responsible vasoconstrictor impulses, originating in the phlebitic segment, can be abolished by sympathectomy or by novocaine ganglion block, resulting in restoration of the peripheral pulsations to normal.

From a practical standpoint, the clinical counterpart of this phenomenon should be encountered often in the treatment of varicose veins where large amounts of sodium morrhuate are used. Actually, it is rare, but it does occur and may be of alarming degree, as illustrated by the following case.

CASE 8 (Roper Hospital No. 118745).—A 53-year-old white man was operated upon Aug. 15, 1941, for extensive varicose veins of the left leg. The main saphenous vein was exposed at the fossa ovalis and just above the knee. The distal segments and several tributaries at each site of exposure were catheterized and injected with a total of 28 c.c. of sodium morrhuate, 5 per cent aqueous solution. Shortly after the patient's return from the operating room, the lower third of the leg and the entire foot became cyanotic and cold, and sensation in the involved area was impaired. The dorsalis pedis artery was palpable. Normal color was

restored by next day after keeping the entire left leg in a heat cradle for several hours. Sympathetic novocaine block was not done but it is now believed that the changes observed in the affected limb were those of severe vasospasm incident to the associated chemical phlebitis.

EDEMA

The presence of edema may complicate the general management of the patient with varicose veins during both the preoperative and postoperative periods. As a preoperative accompaniment of varicosities, edema is a complicating factor mainly from a diagnostic standpoint. It may be of sufficient degree to mask the presence of varicose veins; it may cause a misinterpretation of tourniquet tests by interfering with emptying of distended superficial veins; and it may be due to deep venous obstruction, in which event the presence of varicosities would be the result of processes producing the edema rather than the cause. Under these circumstances, it seems clear that preoperative evaluation of the patient having both varicose veins and edema is incomplete without venographic study of the affected extremity. The routine employment of phlebography under the conditions noted clarifies the circulatory status of the edematous limb and affords a safeguard against saphenous ligation in the presence of femoral venous insufficiency.

The postoperative occurrence of edema in the leg which showed only varicosities before operation constitutes a complication of considerable magnitude. In the presence of partial obstruction or impairment of function of the deep venous system, the sudden obliteration of the saphenous tree by ligation-division and sclerosis results in curtailment of venous return of sufficient degree to cause edema. Although preoperative tourniquet tests are extremely helpful, they are not specific enough to demonstrate subclinical degrees of diminished venous return in the deep circulation. Since such impairment occurs chiefly in women past middle life who have had multiple pregnancies, careful preoperative study of this class of patients is essential and should include venography. Once postoperative edema becomes established, it persists until recanalization of the deep system has occurred. Increase in collateral circulation and promotion of recanalization can be effected by repeated novocaine injections of the lumbar sympathetic ganglia. This form of treatment is recommended as of considerable value, as evidenced by the following case.

CASE 9 (Roper Hospital No. 19046).—A 45-year-old white housewife received the usual ligation-division and sclerosis treatment for bilateral varicose veins in May, 1943. A good sclerosis was obtained and the varicosities rapidly disappeared. However, progressively, increasing edema of the right leg developed after operation and could be controlled only by bed rest. In August, 1943, two paravertebral sympathetic ganglion blocks with novocaine were done twenty-four hours apart. Edema subsided quickly and recurred only to a slight degree after several days of activity. It gradually decreased in extent and finally disappeared completely after one month.

PERSISTENCE OF VARICOSITIES AFTER OPERATION

As previously stated, the objective in the treatment of varicose veins is as nearly complete obliteration of the saphenous tree as possible. If the ligation-division and injection technique is employed, this can be accomplished only by extensive sclerosis which results from the use of large quantities of sclerosant introduced by ureteral catheter in a segmental manner. The chemical thrombophlebitis which follows injection of 20 c.c. or more of 5 per cent aqueous solution of sodium morrhuate is widespread to the extent of being incapacitating. For this reason, hospitalization is advised until the patient's discomfort has diminished. It is believed that the average patient is more willing to submit to a few days of hospitalization than to ambulatory operative methods involving many office injections after operation. Massive sclerosis can be accomplished only by segmental catheter injection and should be done only in hospitalized patients. It represents the principal means of materially reducing the number of follow-up office injections which are both time consuming and disagreeable to the patient.

The majority of patients with varicose veins need no more than one or two postoperative sclerosing injections, so thorough is the obliteration obtained by the massive sclerosis method. A few, however, will show persistence of several veins which must be dealt with one at a time. This is true of individuals having unusual degrees of tortuosity or other anatomic variations which interfere with catheterization at operation. In order to minimize the number of postoperative visits and obtain a maximum degree of sclerosis, injection should be done at the lowest level possible with the extremity elevated, as advocated by Bellis and Churney.[17] It was pointed out earlier in this discussion that sclerosing agents tend to become fixed very rapidly, especially in the presence of a static column of blood. By elevating the leg, the veins are emptied and the sclerosant is allowed to diffuse over a greater surface of intima and thus produce more extensive thrombosis.

REACTIONS TO SCLEROSING AGENTS

While the ligation-injection treatment of varicose veins has been established as a safe and effective method of therapy, undesirable reactions occasionally follow the use of certain sclerosing agents, notably sodium morrhuate. Anaphylactoid responses of extremely severe character have been reported in a few instances after the use of the latter drug.[18-21] Although the mechanism of the reaction is obscure, it seems clear that it is prone to occur upon the resumption of injection treatment in patients who have remained untreated for several months after an initial course of sclerosing therapy, suggesting a sensitization phenomenon. As noted by Prioleau,[22] a minor reaction or a lapse of time between injections should serve as a warning to adopt a different sclerosing agent if further injection treatment is necessary. By em-

ploying the massive sclerosis method of ligation-division and catheterization at the outset, very few follow-up injections are necessary and the dangerous time interval between treatments can be eliminated.

Severe manifestations of an unfavorable reaction to sodium morrhuate are essentially anaphylactic in nature, consisting of angioneurotic edema, asthmatic dyspnea, and peripheral circulatory collapse. I have not encountered such a response during several years of routine use of sodium morrhuate in a representative number of patients. Two toxic reactions have occurred, however, one of a mild and transient nature, the other an extremely severe systemic response of unusual character.

CASE 10 (Roper Hospital No. 20175).—A 26-year-old white man was subjected to bilateral saphenous ligation-division, multiple venous catheterizations, and massive sclerosis with sodium morrhuate in June, 1943. A total of 50 c.c. of the sclerosant was injected through the catheters in the two legs. On the second postoperative day, 5 c.c. of sodium morrhuate were injected into the right main saphenous vein at the ankle and on the seventh postoperative day, 4 c.c. were injected into a varix below the right knee. The left leg did not require further injections. The postoperative course was uneventful and the patient left the hospital showing a satisfactory degree of thrombosis.

One month later, examination revealed complete disappearance of all varicosities except for a small patch in the region of the right knee. Accordingly, 5 c.c. of sodium morrhuate were injected into the remaining veins with the leg elevated. Thirty minutes after the injection, the patient developed a mild headache which was accompanied by hemianopsia. Within two hours, the reaction had reached the peak of its intensity and consisted of severe headache, hemianopsia, and vertigo. The visual disturbance was transient, disappearing and recurring at intervals. The entire syndrome subsided within five hours, leaving no ill effects.

CASE 11 (Roper Hospital No. 33958).—A 66-year-old white farmer entered the hospital in November, 1944, presenting extensive varicose veins of both legs. Examination revealed an unusually severe degree of varicosity of each saphenous system, sacculation and pooling being marked in both thighs as well as in both legs. Scars of previous ulceration were noted about the ankles but no active ulcers were present. Trendelenburg and Perthes tests were positive and there was no past history suggestive of deep venous obstruction. The patient's blood pressure was 155/100. Total white blood count was 5,950 with normal differential picture; red blood count, 4,295,000; hemoglobin, 12 Gm. The urinalysis was normal and the blood urea nitrogen level was 11 mg. per cent.

Because of the widespread extent of the varicosities, two operative procedures were done three days apart. The left leg was treated first in the routine manner by multiple ligations, catheterizations, and injections. The main saphenous vein and its principal tributaries were exposed through five separate incisions, and a total of 25 c.c. of sodium morrhuate was injected through the catheters. No untoward reaction occurred. The immediate postoperative course was satisfactory and the resultant widespread chemical thrombophlebitis was considered adequate.

Operative treatment of the right leg was done after an interval of three days. Because of the remarkable nature of the patient's reaction, the procedure is described in some detail. Local infiltration anesthesia of 1 per cent novocaine was used. Through a transverse incision over the fossa ovalis (Fig. 3), the saphenofemoral junction was exposed. Four tributaries of the great saphenous vein were

ligated and divided; none was injected. The saphenous vein itself was then divided close to the femoral junction and its proximal stump was ligated and transfixed. A two-inch portion of the vein was excised and the distal segment was then catheterized with a No. 5 ureteral catheter. The latter was passed downward a distance of 25 cm. without obstruction, but could not be palpated anywhere in the subcutaneous tissues of either the thigh or leg. Because of failure to demonstrate the catheter within the superficial venous system, it was withdrawn without injection and the wound was packed open temporarily with saline sponges. A large plexus of veins occupying the entire anterior surface of the leg was next isolated between incisions at the ankle and in the upper portion of the calf. Catheterization and injection were done, using 7 c.c. of sodium morrhuate, and the wounds were sutured. There was no reaction. The next step was reexposure of the great saphenous vein just above the knee (Fig. 3). After dividing the vessel, the usual procedure was reversed and the catheter was passed upward toward the groin. Although no obstruction was encountered, the catheter failed to appear at the groin wound and still could not be palpated in the subcutaneous tissues of the thigh. It was withdrawn again without injection and the divided ends of the vessel were ligated. After closure of the supracondylar incision, a final attempt was made to catheterize the great saphenous vein from above. Its downward passage was unobstructed but it could not be palpated through the skin. Despite failure to demonstrate the location of the catheter by palpation, injection of sodium morrhuate was undertaken. When the catheter had been passed 20 cm. from the fossa ovalis, it was withdrawn slowly upward as 5 c.c. of sodium morrhuate were injected through it. Immediately upon completion of the injection, a severe systemic reaction occurred. The patient screamed out because of intense pain located in the midline of the lumbar area of his back. The discomfort developed suddenly and remained confined to its original location without radiation. A hard, shaking chill developed and increased the intensity of the back pain. One-sixth grain of morphine sulfate was given subcutaneously. The patient was removed from the operating table within twelve minutes of the onset of the backache, at which time his pulse was 160 and blood pressure 160/120. Upon reaching his room, he showed signs of mental confusion and the blood pressure fell to 100/70. Within twenty minutes of the onset, the patient was completely disoriented, although conscious, and his blood pressure was 70/40. At this time, 750 c.c. of 5 per cent glucose in normal saline solution were given intravenously, followed by a transfusion of 500 c.c. of whole blood. Within three hours of the onset, the blood pressure was 100/70, but the patient remained confused and disoriented. The axillary temperature was 103° F. Seven hours after the onset, the blood pressure again fell precipitously to 70/50 and the pulse became weak, rapid, and thready. There was still no loss of consciousness and 500 c.c. of plasma were given intravenously; the blood pressure gradually returned to a satisfactory level and was sustained thereafter around 110/75. After ten hours, the patient was mentally clear and his backache had subsided.

During the twenty-four hours after the onset of the reaction, the urinary output was only 210 c.c., while the combined intravenous and oral intake of fluid was 2,330 c.c. The oliguria was transient and a satisfactory output established on the first postoperative day was maintained thereafter. Despite this, the blood urea nitrogen level reached 55 mg. per cent, creatinine 3.82 mg. per cent on the second postoperative day, and returned to normal only after ten days. Repeated urinalyses showed low specific gravity, 2 plus albumin, 2 to 4 pus cells, 1 to 3 red blood cells, and numerous coarse granular casts per high-power field.

The patient was discharged from the hospital sixteen days after the second operation. At that time, it was thought that an excellent result had been obtained, only a few scattered varicose veins remaining. There were apparently no serious consequences of the reaction.

Comment.—The reaction noted in Case 11 was, in all probability, not due to hypersensitivity to sodium morrhuate. This is probably true for three reasons. First, the time interval between injections was only three days; second, injection of several veins below the knee was carried out prior to the thigh injection without a reaction; third, the clinical manifestations were not those of an allergic reaction, that is, there was no urticaria, asthmatic dyspnea, or mucous membrane exudation. The general systemic response was undoubtedly initiated by the injection of the sodium morrhuate into the general circulation by way of the femoral vein. Inasmuch as the catheter could not be palpated in the subcutaneous tissues, one can assume that it had entered the femoral vein through a large communicating vessel. This is especially likely in view of the free, unobstructed progress of the catheter as it was passed along the vein. The fact that there was no subsequent evidence of femoral obstruction (due to sclerosis) does not mitigate the latter possibility, because sclerosis from sodium morrhuate occurs only in the presence of venous stasis. If the foregoing reasoning is correct, the reaction can be considered as a specific response of the general organism to a large amount of sodium morrhuate delivered directly into the systemic circulation.

SUMMARY

1. Radical surgical treatment of varicose veins by high ligation-division, catheterization, and massive segmental sclerosis is recommended as the method of choice in obtaining a large percentage of satisfactory results.

2. Factors which tend to complicate the treatment of varicose veins are discussed and illustrative case reports are presented under the headings of anatomic variations, thrombophlebitis, cellulitis, ulcer, postoperative vasospasm, edema, persistence of varicosities after operation, and reactions to sclerosing agents.

3. The interruption of sympathetic nerve impulses as an adjunct in managing certain complications is discussed.

4. A case report of an unusually severe reaction to sodium morrhuate is presented in detail.

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CLINICAL ANATOMY OF THE VERTEBRAL VEINS

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THE various explanations offered for the spread of so-called "paradoxical metastasis" have, until recently, been unsatisfactory. Fig. 1 illustrates such a case and shows metastasis to the cervical spine from carcinoma of the prostate, without x-ray evidence of metastasis to the lung. How do such metastases reach the cervical vertebra through the usually accepted avenues of spread? Likewise, metastasis to the spine can be found in early carcinoma of the breast without evidence of lymph node metastasis. The frequency with which suppurative or malignant processes in the thorax produce intracranial lesions is such as to point to anatomic structures conveying these organisms or cells to the cranium.

Everyone is familiar with the usual mode of spread of infection or malignancy. The first is by lymphatics, where the organism or tumor cell migrates into the lymphatic channels and is transported to the adjacent lymph nodes where it is filtered out. Eventually, if the bacteria or tumor cells reach the thoracic duct, they will finally enter the venous circulation which leads directly to the right side of the heart and then to the lungs. That such an organism or cell can filter through the tissues of the lung, without setting up a local focus in the lungs, is highly improbable. Therefore, one would expect to find definite evidence of metastasis to the lungs before finding metastasis to other parts of the body, such as the spine.

The second mode of distribution of bacteria or tumor cells is by way of the veins. In this case, bacteria or tumor cells infiltrate into venous channels by erosion of the original lesion into such channels. In such instances, the spread would be along the same route, namely, into the caval system to the right heart and to the lungs.

Finally, spread of the disease is sometimes carried by the arterial system directly. In such cases, the bacteria or cells enter the arterial system by erosion of the original lesion into the pulmonary veins, then to the left side of the heart to the systemic arterial circulation. A typical example of this is miliary tuberculosis.

To explain the so-called "paradoxical metastasis" different theories, supported in some instances by careful work, have been advanced. One is that in some patients there is a patent foramen ovale, and that the bacteria or tumor cell finds its way to the left side of the heart through the foramen ovale and is then spread by the arterial system. In such event, the lodging of the cell or embolus would be purely accidental. This theory is traced to the two cases published by Litten,[1] 1880, and Cohnheim,[2] 1889, in which the foramen ovale was patent. Since then,

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this theory has been copied in textbooks of pathology, and credence has been given to this theory, although Cohnheim stated that, "the locality to which the detached thrombi are transported is determined by anatomical conditions alone."

Another theory is that advocated by Warren, Harris, and Graves[3] who state: "While hematogenous metastasis may occur in carcinoma of the prostate, the chief factors in the marked preponderance of metastatic lesions in the pelvis and in the lower regions of the spine are the distribution of the nerves and the condition of the tumor cells along the perineural lymphatic spaces, and the close contact with the bone." The perineural lymphatic dissemination of the tumor may be either by continuity or through embolism. No doubt local metastasis occurs in this manner at times. But these authors do not attempt to explain distant metastasis of the prostate such as shown in Fig. 1.

Fig. 14.—Showing extensive metastasis to the cervical vertebra from carcinoma of the prostate.

A different explanation offered is that tumor cells have affinity for certain tissues; thus, carcinoma of the prostate is said to have predilection for metastasis to bone and carcinoma of the lung selects brain tissue when it is separated from its mother lesion.

Finally, one theory is based upon careful histologic studies of the lungs, where it has been found that tumor cells were present in the lungs without having produced sufficiently large metastasis to be detected by x-ray. This has been found in cases where obvious distant metastasis have been demonstrated. It has been prosposed that the

tumor cells have been transported into the lungs and thence into the left side of the heart without producing growth in the lungs. The objection to this is that it is highly improbable, as lung tissue is a fertile field for the growth of both bacteria and tumor cells. As Batson has pointed out, these findings could be explained more readily as terminal events and as having come from the secondary metastasis.

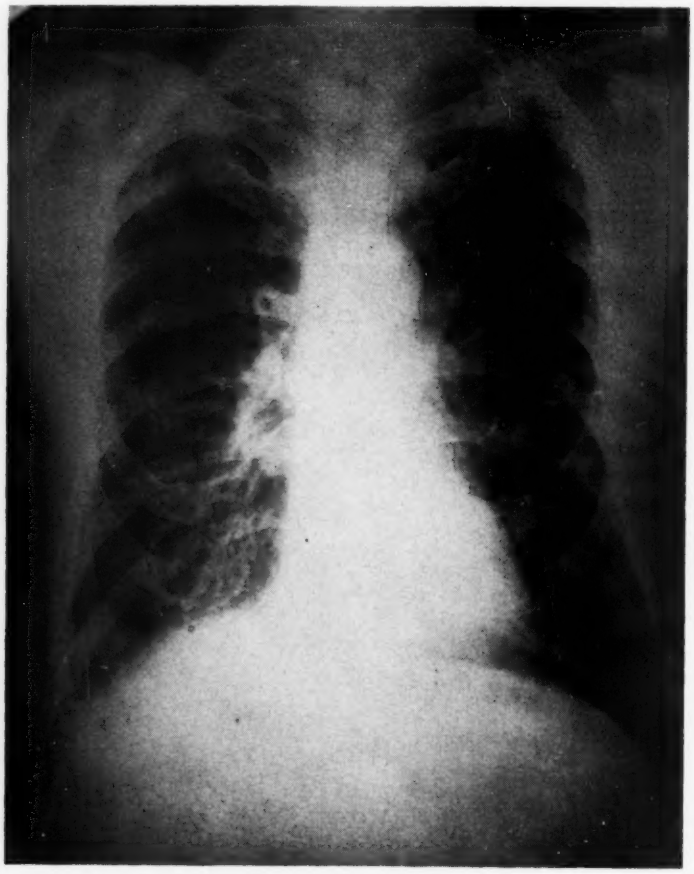

Fig. 1*B*.—Showing no x-ray evidence of metastasis to the lungs. Same case as Fig. 1*A*.

In 1940, Batson[4] published his report on the rediscovery of the vertebral system of veins. While it is true, as Harris[7] has pointed out, that the vertebral system of veins was described and understood over 100 years ago, credit should be given Batson for rediscovering this system of veins, employing the injection method. Standard textbooks of anatomy and even Franklin's[8] *A Monograph on Veins*, 1937, make no mention of this system. Forgotten knowledge is equivalent to no knowledge. Batson's approach is entirely new and demonstrates more clearly the connections between this and other venous systems then could be done by dissection.

In reviewing the history of the early work on the vertebral system of veins, I have drawn freely on the article by Harris.[7] The early anatomists were familiar with the spinal veins and their connections. Bock[9] of Leipzig, in 1823, published a work called *Darstellung der Venen* in which he demonstrated most completely the rich venous plexuses of the inside of the bony spinal canal with dominance of the longitudinal anastomoses between the bony canal and the theca of the dura mater. Bock also pointed out that the sacral branches of the internal iliac veins communicate freely with the veins within the spinal canal through the anterior spinal foramina. The posterior plexus on the back of the sacrum does the same through the posterior sacral foramina. All the communications with the cranial venous sinuses are given in detail. He pictures the transverse anastomosis between the two longitudinal channels and describes a "circellus venosus" which is situated on the posterior aspect of the body of each vertebra. These receive tributaries from the marrow cavity of the vertebra. Willis[10] in 1664, Winslow[11] in 1732, Breschet[12] in 1832, and Cruveilhier[13] in 1834, all knew about the spinal veins.

SUMMARY OF BATSON'S WORK

In his first experiment, Batson injected the dorsal vein of the penis with a fairly thick solution of King's yellow artist's water color, and found that the material entered the smaller veins of the pelvis along the prostate, thence to the iliac vein, and into the vena cava. When a thin solution of Weber's artist's water color vermilion was used, the material went into the veins of the ileum, then it progressed to the spine. Some of the intercostal veins were filled, and after he had injected a total of 200 c.c., the material was found at the base of the skull and in the cranial cavity. The substance also went distally to the vessels of the right thigh where it was found that these vessels were the vena vasorum of the femoral vessels. None of the material entered the vena cava. He then injected the dorsal vein of the penis of the macacus rhesus monkey with colloidal thorium dioxide. The material was found to go into the pelvic veins and ascend to the inferior vena cava. When pressure was made on the inferior vena cava, the material entered the vertebral system of veins as well as the vena cava.

When the small veins of the female breast were injected with this solution, it was found that it spread to the clavicles, to the intercostal veins, to the head of the humerus, to the cervical vertebra, and to the transverse and superior longitudinal sinuses of the skull. It also entered the azygos vein and the superior caval system. He then injected the small veins adjacent to the nipple and found that the material spread to the tissues immediately adjacent to the point of injection, and sometimes to the opposite breast as well as more distally to the scapula and humerus.

Batson's report offered an anatomic explanation for "paradoxical metastasis." Because of its great interest and practical importance, the experiments have been repeated. The dorsal vein of the penis was injected with Weber's artist's water color vermilion. By taking serial x-rays of the body, it was demonstrated that the injected mass flowed through veins along the pelvic girdle as far distant as the head of the femur, along the vertebral column, and finally inside the cranium, without entering the vena cava. Five cadavers were used, and in four the flow was as described; in only one the material entered the vena cava and did not proceed up the vertebral column. Unfortunately, due to lack of suitable material, I was unable to repeat his work on the injection of the female breast.

Fig. 24.—Showing cannula in dorsal vein of penis. The material is shown ascending into the vertebral veins.

DISCUSSION

Once a carcinoma cell or bacterial embolus has been ushered into the vertebral system of veins, how can it reach distal structures against the usual direction of flow of blood? Thus, how does cancer of the breast produce metastasis to the lumbar vertebra or to the proximal end of the femur if the tumor cell would have to flow "upstream" as it were? Likewise, how does a bronchogenic carcinoma cell reach the vertebral system,

and from there, how is it transported inside the cranium against the normal flow of venous blood? Batson[4-6] states that reversal of flow in the vertebral system of veins does occur under certain circumstances such as sneezing, coughing, or straining. Thus, when a person is in the upright position, anything that increases the pressure within the body cavities would predispose to reversal of flow, with gravity. Likewise, when in the prone position, reversal of flow to the head would be facilitated. Batson also found that the pressure in the vertebral system of veins is low, considerably lower than in the caval system. Also, the spinal veins have no valves, a factor which is of great importance in the reversal of flow. On this subject, John Hilton,[14] 1855, stated: "The

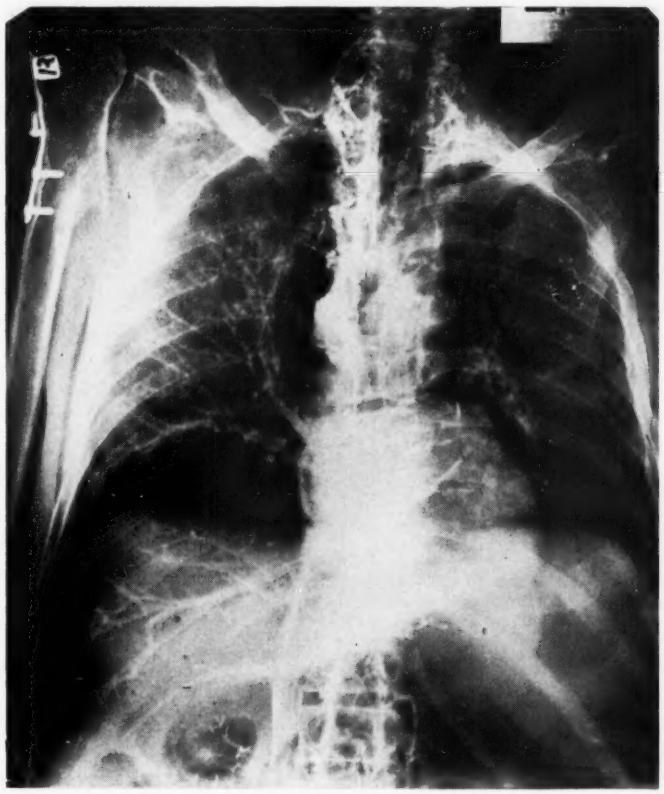

Fig. 2*B*.—Showing the material ascending in the dorsal and cervical region without being present in the inferior vena cava.

absence of valves in the whole of these venous tubes is a circumstance which is doubtless connected with a wise intention. It enables the blood to pass in either direction, and consequently, greatly increases the freedom of venous circulation, a point of essential importance with an organ whose functional capacity is so liable to interruption under so slight a disturbance of the balance of its circulating fluid."

What further evidence is there to support reversal of flow of fluids in the body? According to Franklin's[8] monograph on veins, the summary

of the findings of different workers measuring venous pressures shows generally that the pressure in the more distant veins is higher, up to plus 11 or 12 mm. of mercury, and as the venous blood approaches the superior vena cava, it may be minus 2.8 mm. of mercury. It is also minus in the external jugular vein. According to the same author, Ledderhose,[8] in 1906, pointed out that there can be a backward wave of blood in veins without backward movement of blood. Retrograde movement of blood is said to occur at the site of the venous valves. Naegeli

Fig. 2C.—Showing material in the vertebral veins of the cervical region entering the cranium.

and Janker[8] performed experiments on embolism. Under x-ray, emboli were injected into the femoral vein, passed up the abdominal vena cava, stayed for a while near the diaphragm, and then shot along the thoracic inferior vena cava to the right auricle. Thereafter they often came back again to the diaphragm before going through the heart. Franklin[8] states: "Under pathological conditions of the venous system in man, change of posture must at times result in temporary reversal of flow in veins." Other workers have noted backward flow of blood in the veins. Certain evidence of backward flow of fluids in the body is pres-

ented by Hamilton, Woodbury, and Harper.[15] These workers experimented with a monometer of their own construction, measuring the effect in the arterial system of sneezing, coughing and straining. There was definite, and at times, rather marked increase in the systolic and diastolic pressures, sometimes up to 100 mm. of mercury. They also measured the intraspinal pressure and found it to rise markedly during coughing and sneezing. No measure was made of the venous pressure. But it can be inferred from the experiments that there is also a rise in the venous pressures.

Now, as to the pathways through which a bronchogenic carcinoma cell can be transported into the vertebral system and thence to the cranial cavity, Lee,[16] in 1922, performed experiments ligating the thoracic duct. His summary is as follows: "It seems that the integrity of the thoracic duct is not essential to the life of the animal. In some cases in which the ligation was absolute, collateral lymph circulation was established to the right thoracic duct; while in other cases which showed complete ligation, *lymphaticovenous connections were found to exist between the thoracic duct and the azygos vein.*" Batson demonstrated connection between the azygos and vertebral system of veins.

It is quite conceivable that metastatic bronchogenic carcinoma cells can be transported into the vertebral system, either through the lymphatics of the lung emptying into the lymphatic system, or by retrograde flow into the azygos system, and thence into the vertebral system. It is also possible that such cells might enter bronchial veins, thence into the azygos veins and into the vertebral system.

Clinical application of the vertebral system of veins as channels, through which infected emboli are transported, has been made by Martin.[17] He reports three cases of superior sinus thrombosis following childbirth, with one fatality.

From the forgoing experiments, one is justified in concluding, that there is reversal of flow, not only in the vertebral system of veins, but also in other veins as well as the lymphatic channels.

THE VERTEBRAL VEINS—A DETOUR FOR BLOOD

Recent work on ligation of large venous channels for thrombosis and embolism, such as the femoral, iliac, or even the inferior vena cava itself, has re-emphasized the part that the vertebral system of veins play in returning blood from the lower extremities. This also, according to Harris, was known over 100 years ago. Cruveilhier,[13] 1834, wrote: "The veins of the spine are such that one may consider them, in relation to the general circulation, as establishing an uninterrupted communication between the veins of all parts of the trunk: so that one may conceive either of the two venae cavae to be obliterated, without interruption of the venous circulation. Even the vena azygos major, usually considered as the main route of communication between the superior and inferior

vena cava, is not necessary, in view of the presence of the anterior and posterior rachidian plexus. This explains why I have seen at one time the inferior vena cava, at another the superior vena cava obliterated without any visible enlargement in the calibre of the azygos vein, and which may seem surprising, without any edema either of the upper or lower limbs.''

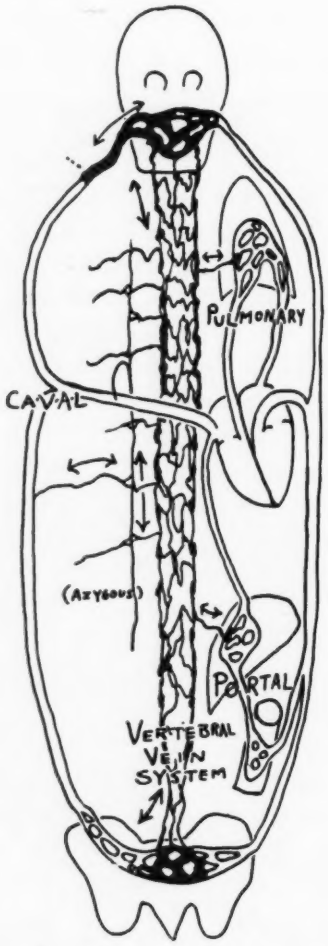

Fig. 3.—Schematic representation of the vertebral system and connections with other venous systems. (From Batson, Oscar V.: Ann. Surg. 112: 138, 1940.)

SUMMARY AND CONCLUSION

Injection experiments, according to the technique of Batson, were performed on cadavers by injecting the dorsal vein of the penis. Results similar to those reported by Batson were obtained. It is therefore concluded, with Batson, Bock, Cruveilhier, and others, that there is a fourth system of veins, namely, the vertebral or meningorachidian system in addition to the caval, portal, and pulmonary systems, through which tumor cells or infected emboli are sometimes spread to distant parts; that these veins are of the primitive type, having no valves and carry-

ing the blood at low pressures which enables reversal of flow under certain circumstances, such as posture, coughing, sneezing, and straining; that this system of veins furnishes an anatomic explanation for the so-called "paradoxical metastasis." It is further concluded that the vertebral system of veins furnishes channels through which blood returns to the heart when large venous structures such as the femoral or iliac veins are occluded.

I wish to thank Dr. Robert Stone Dow, professor of Anatomy, University of Oregon Medical School, Portland, Oreg., for valuable suggestions in preparation of this paper. Also, I wish to thank the King County Hospital, Seattle, for making material available for this study.

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HOMOGENOUS CARTILAGE GRAFTS

An Experimental Study

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As I have previously reported there is some confusion regarding the viability of various types of cartilage transplants. Because of varying opinions and so-called "refrigerated isografts" I undertook an experimental study of autogenous cartilage grafts in dogs.[1] From that experiment, which was in agreement with previous experimental evidence and clinical impressions, it appeared that autogenous costal cartilage, when transplanted to the subcutaneous tissues in dogs, remained viable and did not lose weight or volume up to as long as one and one-half years. Whether the cartilage was transplanted with or without perichondrium did not alter the facts.

Since that time I have secured another bit of human experimental evidence which completely corroborates that experiment. A woman came in for advice concerning an asymmetry of the nose. Twelve years before, an autogenous costal cartilage graft had been used to correct a traumatic saddle-nose deformity. In correcting the asymmetry it was necessary to remove the previous graft for reshaping and a portion was saved for miscroscopic section. This showed normal appearing hyaline cartilage.

I think one can safely state that the evidence is incontrovertible that autogenous hyaline cartilage grafts retain normal gross and microscopic appearance for long periods. Moreover, degenerative changes are but little, if at all, accelerated by transplantation.

On the other hand, the data concerning the survival of homogenous cartilage grafts is quite meager. There are a few clinical reports. In 1937 Gillies[2] attempted and reported the successful transplantation of the cartilage framework of an ear from a mother to her son. J. B. Brown,[3] in 1940, reported clinically successful cases of rib cartilage transplants from father and mother to sons, in the correction of traumatic facial deformities. However, since specimens have not been removed later for examination, one has no proof that these transplants do not actually die and remain merely as foreign bodies.

The experimental evidence is conflicting. Loeb and Siebert,[4, 5] in the course of studies concerned with the transplantation of various tissues, state that there is greater tissue reaction to, and more eventual absorption of, homogenous cartilage grafts than of autogenous.

Kirkham[6] found that auricular cartilage removed from one rabbit and placed in the abdominal subcutaneous tissues of another retained

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its size and form but on section the cell spaces were vacuolated and nuclei absent. It should be stated that these experiments were an attempt to discover how long after death a homogenous cartilage transplant was possible. A rabbit was killed and one hour later a piece of ear cartilage was removed and placed in the abdominal wall of a living rabbit. At hourly intervals up to six hours another piece was removed and transplanted to another living rabbit. The transplants were removed in six months and examined.

From this experiment Kirkham concluded that cellular death of the transplants occurred but their form was retained. As will be seen, the experiment here reported disagrees with these findings.

Fig. 1.—Homogenous costal cartilage grafts removed from the abdominal wall of a dog one and one-half years after transplantation. Note the glistening translucent sheen of normal-appearing cartilage.

METHOD OF EXPERIMENT

The same general method was used as in experimental transplantation of autogenous cartilage, except that two dogs were anesthetized and segments of costal cartilage removed from one were transplanted at once to the abdominal wall of the other. Five pairs of dogs were used.

In each instance care was taken to secure cartilage as nearly free from central ossification as possible. This must be kept in mind as central ossification of rib cartilage takes place quite early in dogs. The cartilage was removed through an oblique incision over the costal margins and cut into segments about one and one-half inches long. These were measured and then inserted into the abdominal subcutaneous tissues of the other animal through small incisions just to the right and left of the midline. Six pieces were implanted in the abdominal wall of each animal, three on the right, and three on the left. In this experiment soft tissues were removed from the transplant but no attempt was made to strip off the perichondrium, since in previous work it was

found that the presence or absence of perichondrium had no effect on survival of autogenous cartilage grafts. The various incisions were closed with silk.

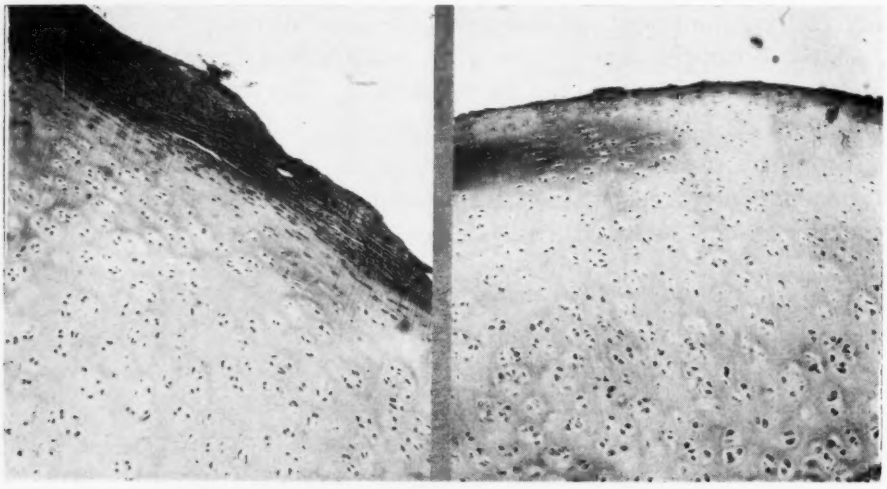

Fig. 2.—Photomicrographs (×100) showing the architecture normally seen in costal cartilage of young adult dog.

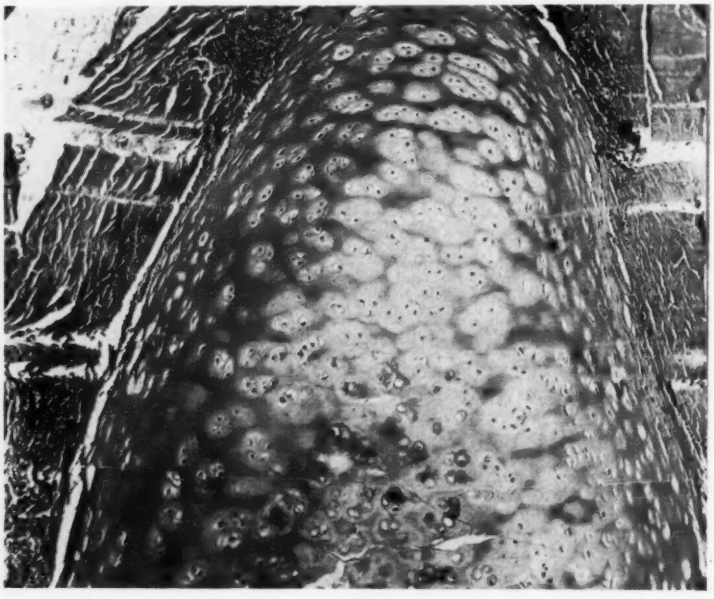

Fig. 3.—Photomicrograph (×100) showing homogenous costal cartilage two weeks after cross transplantation to abdominal subcutaneous tissues.

Thus, sixty pieces of costal cartilage were implanted in the abdominal walls of ten dogs, in each instance the cartilage being obtained from a donor dog. These grafts were then removed from a pair of dogs at intervals. In this way twelve grafts were removed at two weeks, three months, six months, one year, and one and one-half years.

Fig. 4.—Photomicrograph (×100) showing homogenous costal cartilage graft removed after three months in abdominal wall. The cell architecture is normal.

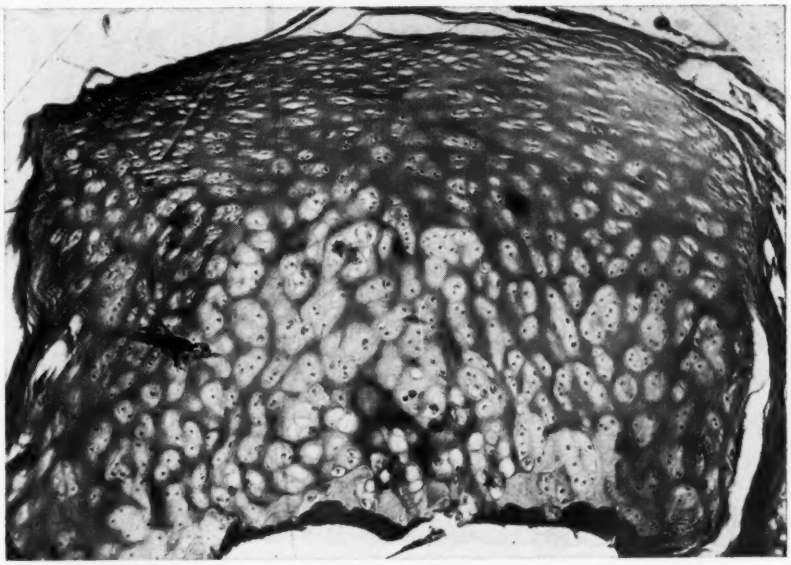

Fig. 5.—Photomicrograph (×100) showing homogenous costal cartilage transplant removed from abdominal wall after six months. There is moderate increase in the clear zones of the cells and a few cells without nuclei.

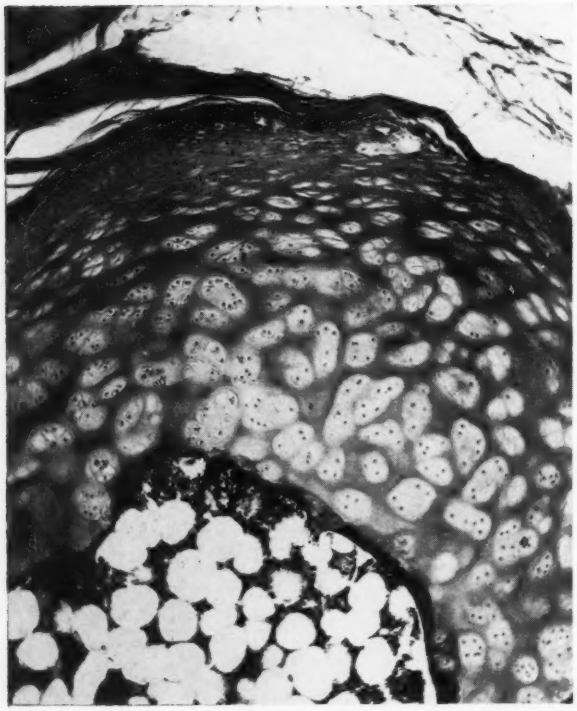

Fig. 6.—Photomicrograph (×100) showing specimen removed after one year in abdominal subcutaneous tissues of recipient dog. The cells are well preserved. A central marrow cavity is present.

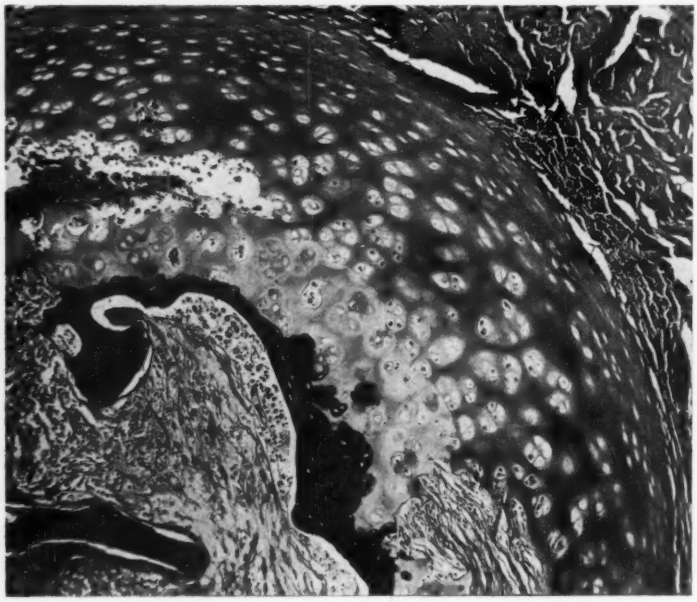

Fig. 7.—Photomicrograph (×100) showing a specimen removed one and one-half years after cross transplantation. This particular one was selected because it showed as marked central bone formation as any examined. Some specimens at one and one-half years showed relatively little.

GROSS EXAMINATION AND MEASUREMENT OF TRANSPLANTS

During the time the grafts were in place in the abdominal wall they could be felt and, in most instances, seen. Of the sixty pieces transplanted, all but two were recovered and examined. These two may not have been absorbed but may have migrated to a spot where they were not discovered, since it was noted that there was a tendency for the grafts gradually to change location.

On removal the transplants were firmly attached to the soft tissues without noticeable inflammatory or fibrotic reaction about them. They were removed by sharp dissection and cleared of soft tissues. They retained their cartilaginous appearance and springlike physical properties. In some instances softened yellowish spots were present which were thought to be areas of degeneration. The grafts were measured and it was found that there was no significant change in size.

All the recovered transplants were sectioned and examined microscopically. The accompanying photomicrographs (Figs. 1 to 7) are representative ones selected at the various time intervals. I believe that if presence of normal cartilaginous architecture is a criterion that one can state without hesitation, these cross-transplants remain viable. It is true that degenerative changes are present in that the clear zones around the nuclei are larger and central ossification is present. However, in interpreting this, one must remember that the ossification of costal cartilage in dogs is an early occurrence, and that in some instances early central ossification was already present when the transplants were carried out.

CONCLUSIONS

From this experiment it appears that costal cartilage can be transplanted from one dog to the subcutaneous tissues of another dog and remain viable in its new location up to one and one-half years.

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TRAUMATIC CHYLOTHORAX

Report of a Fatal Case Complicated by a Ruptured Duodenal Ulcer

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CHYLOTHORAX resulting from rupture of the thoracic duct due to trauma is an unusual condition which presents a challenging therapeutic problem. Dorsey and Morris,[6] in 1943, reviewed fifty-eight cases from the literature and reported one additional case. Doubtless, there are many unreported cases. Prognosis is grave since there is a 50 per cent mortality in cases in which operation has not been done, and direct surgery to repair the duct has been completely unsuccessful, resulting in 100 per cent operative mortality.

Traumatic chylothorax is often associated with fractured rib, fractured vertebra, or dislocation of vertebral bodies. However, it is not uncommon to find chylothorax following nonpenetrating injuries unassociated with any bone or joint injury which is demonstrable by clinical roentgenologic or post-mortem examination.

Frequently there is a history of the patient having fallen or being thrown on the back or having been in an auto accident where there is sudden hyperextension of the spine. The mechanism in such instances is thought to be that of stretching the thoracic duct and because the cisterna chyli is fixed beneath the diaphragm, rupture ensues.[3] Following rupture, chyle escapes retropleurally and, later, the pleura ruptures, causing chylothorax as found at autopsy by Cellan-Jones and Murphy.[4] In 1932, MacNab and Scarlett[14] drained, by thoracotomy, a retropleural cavity which at post mortem was seen to communicate with the pleural cavity. The most frequent site of rupture in nonpenetrating injuries is in the supradiaphragmatic portion in the region of the ninth to tenth thoracic vertebrae, and leakage is usually into the right pleural cavity, since the duct at this level is on the right side and does not cross to the left side until the level of the seventh thoracic vertebra is reached. Of twenty-eight cases reviewed by MacNab and Scarlett,[14] chylothorax was on the right in fifteen, on the left in eight, and on both sides in five. Shackelford and Fisher[17] suggest that rupture may be due to increased intrathoracic pressure, but in discussing their paper Penick stated that valves in the thoracic duct are inefficient and that any extraductal pressure would force fluid in two directions and not increase tension on the duct wall. The former authors[17] also point out that several cases have followed ingestion of a heavy meal, possibly because the intraductal pressure is highest at this time. In

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1939, Everhart and Jacobs[7] reported a case of right chylothorax in a newborn infant, thought to be due to trauma during or after birth. They collected five such cases in infants under 1 year of age and ten cases in those from 1 to 14 years, the etiology being either trauma or rupture secondary to obstruction by tuberculous nodes. Chylothorax caused by nodes were likely pseudochylous rather than true chylothorax. Beatty,[2] in 1936, reported a case of right chylothorax which developed six and one-half years after depressed fracture of the left clavicle. This is the longest reported interval between the trauma and the appearance of the lesion, but there was evidence of deformity of the subclavian vein and the chylothorax was doubtless due to the obstruction rather than direct trauma to the duct, however, both factors may have played a part.

In contradistinction to nonpenetrating injuries in which the lesion is usually on the right side, in penetrating wounds of the thorax the left side is more frequently involved (nine of fourteen, Shackelford and Fisher).[17] Right chylothorax was the result of closed injuries in eighteen cases and open injuries in two cases. It is suggested that an injury low in the thorax results in a right-sided chylothorax and that a high injury causes a left chylothorax.[17] Watts[21] reported a case of self-inflicted knife wound of the suprasternal notch which severed the thoracic duct (proved at autopsy) and resulted in left chylothorax. Bullet wounds also have resulted in severance of the thoracic duct. Matson and Stacy[15] reported a bullet wound of the duct successfully treated. The thoracic duct also has been injured surgically. Smith and Woliver[18] report a case which followed resection of the right tenth rib for sarcoma. Turbid fluid was noticed in the wound near the end of the operation, the duct was not seen or ligated, several thoracenteses were done, and four intravenous injections of the fluid were given; the patient survived.

Clinically, in the patient with chylothorax there is a relatively asymptomatic interval between the trauma and onset of marked dyspnea, pallor, cyanosis, thready pulse, and shock. This period averages four days,[14] during which time the patient's only complaint may be mild chest pain. The longest time interval is that reported by Beatty,[2] of six and one-half years. Usually following thoracentesis, the dyspnea is relieved, the cyanosis disappears, and the shock is relieved. However, there is a rapid reaccumulation of the fluid and aspiration must be repeated, sometimes within twelve hours. There is rapidly progressive loss of weight and emaciation. Physical examination at the onset reveals a dyspneic patient with weak pulse and low blood pressure; there is evidence of fluid in the pleural cavity. These findings are present each time the fluid reaccumulates.

A definite diagnosis is made by examination of the fluid which is milky in appearance. However, the first thoracentesis may yield milky

fluid with a pink tinge. Rapid accumulation helps to distinguish it from pseudochylous fluid, as well as the fact that true chyle forms a creamy layer on top when allowed to stand. Fine fat globules are seen microscopically when stained with sudan III. The degree of opalescence is constant on repeated tappings, whereas opacity of pseudochylous fluid diminishes. Specific gravity of true chylous fluid is over 1.012 and that of pseudochylous fluid is less than 1.012. The total solid of chylous fluid is about 4 per cent and total protein is 3 Gm. per cent, while total solid of pseudochylous fluid is less than 2 per cent, and total protein is 1 to 3 Gm. per cent.[20] The albumin content is greater than that of globulin in chyle: a ratio of 3.68 to 1.[5] Total fat content of true chyle is 0.4 to 4. per cent and that of pseudochyle is very low, averaging 0.3 per cent.[14] During the patient's illness there is a progressive decrease in the total protein content of the blood. Repeated aspirations of the pleural cavity are necessary to relieve the patient's dyspnea and shock. Generally as soon as the procedure is completed the symptoms are relieved. Supportive intravenous amino acids, plasma, and blood are necessary but frequently ineffective in replacing lost plasma protein. The dietary therapy varies from nothing by mouth to a fat-free diet. In the case presented here, the fluid became clear yellow following an oral intake limited to water. No similar observation has been reported. Lillie and Fox[13] report a case of recovery in which the only therapy was fat-free diet.

Bauersfeld,[1] in 1937, reported the first chylothorax to be successfully healed by the intravenous administration of the aspirated chyle. In this case, 22,275 c.c. of chyle were aspirated and 1,200 c.c. were given intravenously. He stated that he believes sealing of the duct was incidental with administration of the chyle. In 1943, Schnug and Ransohoff[16] reported eight patients who had been treated by the intravenous injection of the aspirated chyle. Three of these patients recovered, three died, and two were "unchanged." Whitcomb and Scoville[22] reported a fatality following the cautious intravenous administration of only 75 c.c. of chyle. They considered death to be due to anaphylaxis. They emphasize the necessity of culturing and of determining a possible sensitivity to the chyle before it is administered intravenously. They believe that if on microscopic examination fat globules of 3 micron diameter or larger are found, then chyle should not be given intravenously. Little, Harrison, and Blalock[12] showed that total lipids are markedly increased after chyle is given intravenously, but that after five and one-half hours the lipids return to their previous level; the phosphatids remain elevated however. Schnug and Ransohoff[16] reported a marked "phlebothrombosis" which made intravenous administration difficult. This has been noticed by others and was an early finding in our case, it being necessary to expose veins surgically several times. This intravenous clot was associated with changes in

the vein wall and was a thrombophlebitis in the case reported here. The mechanism of this phenomenon is not definitely explained but is probably due to the hypertonicity of the fluid. A satisfactory solution would enhance the therapy and change greatly the prognosis in these cases. In 1938, Johnson and Freeman[11] presented evidence that there is some hemolytic agent in chyle. Samples of blood with lacteal and thoracic duct lymph showed slight hemolysis. They[11] also showed that lacteal lymph destroyed twice as many red blood cells as does thoracic duct lymph. They[11] suggest that glycerol may be the hemolytic substance. In 1940, the same investigators[8] state that only thoracic duct lymph has the property, other lymph not being hemolytic. Soap and fatty acids[8] are present in sufficient quantities to account for the hemolytic action. Duct lymph[8] of fasting dogs contains too little fatty acids or soap to produce hemolysis. Since, in severance or rupture of the thoracic duct, the chyle does not reach the blood stream, it appears logical to us to conclude that the rapid and frequent intravascular clotting may be explained on the fact that this hemolytic substance is kept from the blood stream leaving an imbalance in the compensatory mechanism. Since Freeman and Johnson[8] have suggested that soap and fatty acids may be the hemolytic agents, intravenous administration of fats may prevent this clotting phenomenon as well as serve as an intravenous nutritional substitute. In 1935, Holt, Tidwell, and Scott[10] reported intravenous use of fats in sixteen infants with severe nutritional disorders; the fatal outcomes could not be attributed to the fats, but neither could the good results. The dosage used in this series was 1 to 2 Gm. per kilogram body weight. The difficulties in administering fats intravenously are in obtaining an emulsion stable enough to resist sterilization and the possible danger of fat embolism. Four of Holt, Tidwell, and Scott's patients[10] came to autopsy within forty-eight hours after injection and in all there was adequate explanation of the death besides the intravenous fat; a fatty liver was found in two of the four patients, which is more frequent than occurs in these cases without intravenous administration of fat. Use of intravenous fat in cases of traumatic chylothorax is suggested as a caloric source and as a possible prevention of intravascular clotting. Heppner,[9] in 1934, and Brown,[3] in 1937, reported the same case of traumatic chylothorax treated by external drainage of the posterior mediastinum, which was a cavity found to communicate with the pleural cavity. The patient died the following day while at stool. At autopsy bilateral adrenal apoplexy was found.

Fat-free diet has been used in several cases, but there has been little to no reduction in the amount of fluid produced. In our case the character of the fluid aspirated changed from creamy to straw color when the patient was put on fat-free diet. This has not been previously reported.

Phrenic crushing has been used in an attempt to close the opening in the duct, the rationale of its use being that sealing of the opening is favored by causing an elevation of the diaphragm. In our case, in addition to this procedure, pneumoperitoneum was done for the same purpose. This is the first time the latter procedure has been used. Schnug and Ransohoff[16] injected broth intrapleurally to stimulate pleural reaction in their case in a hope to occlude the lumen in the thoracic duct. The patient had complete cessation of fluid collection, but since there was no polymorphonuclear or temperature response they questioned the efficacy of the broth.

The case we are presenting is typical and includes an unreported autopsy finding.

CASE REPORT

A. O., 14 years of age, white male, was admitted to Charity Hospital Jan. 28, 1944, and died Feb. 18, 1944. His chief complaint was pain in the right side of the chest and shortness of breath. On January 24, the patient fell from an iron framework, striking the right thorax. He developed constant pain in the chest, but did not consult a physician until the next day. There was no evidence of fractured ribs or other injury and the patient was sent home. The pain continued to increase and the patient became dyspneic, Jan. 28, 1944, at which time he returned to his doctor who referred him to Charity Hospital. There was no history of hemoptysis. The patient could lie only on the right side because of the shortness of breath while he was in other positions. On admission, blood pressure was 120/70; temperature, 98.4° F.; respiration, 30; and pulse, 100. The patient was very dyspneic and stayed on the right side most of the time. Physical examination revealed dullness over the entire right hemithorax. Roentgen examination showed clouding of this area. Eleven hundred cubic centimeters of pink creamy fluid were aspirated from the right chest. The fluid did not clot. Thoracentesis was repeated whenever necessary for dyspnea and the accompanying picture of shock. This had to be done approximately every eighteen to twenty-four hours and a total of 26,855 c.c. of fluid was removed. The temperature never exceeded 100° F. and was usually normal except on the last day, when it rose to 104.2° F. (rectal). Pulse and respiration varied with the accumulation and withdrawal of fluid. With each accumulation the patient would become dyspneic, the blood pressure drop, and pulse rate become rapid and weak. On admission the patient was a healthy boy, but soon became thin and emaciated. His stated weight at home was 120 pounds, autopsy weight was 97 pounds, and Feb. 4, 1944, he weighed 108 pounds. Feb. 5, 1944, a fat-free diet was started and Feb. 10, 1944, the patient was allowed only water by mouth. Feb. 11, 1944, the character of the fluid changed from creamy consistency to slightly cloudy straw-colored fluid. Feb. 9, 1944, a right phrenic crushing was done under local anesthesia. Feb. 14, 1944, a pneumoperitoneum, injecting 1,100 c.c. of air, was done. The patient received 2,700 c.c. of blood, 1,400 c.c. amino acids, and 2,300 c.c. chyle, intravenously. Infusions were difficult because of the tendency toward intravenous clotting. The sterility of and lack of sensitivity to the chyle was determined before each intravenous administration. There were no apparent deleterious effects of the intravenous administration of the chyle. Jan. 29, 1944, the hemoglobin was 85 per cent, red blood count, 4,900,000; white blood count, 13,920, with 78 per cent polymorphonuclears, 18 per cent lymphocytes, and 4 per cent monocytes. Urinalysis was essentially negative. Serum protein dropped from 5.11 on Feb. 3, 1944, to 4.42 on Feb. 7, 1944. Total protein content of the chyle on two occasions was 2.29 and 2.0. Gastric analysis, Feb. 7,

1944: Fasting free HCl was 10 degrees and total HCl was 15 degrees; thirty minutes after histamine, fasting was 55 and total was 81. The patient's progress was steadily downhill with increasing emaciation, loss of weight, and apathy. Feb. 12, 1944, he vomited dark brown feculent material. Feb. 15, 1944, he complained of burning in his throat and stomach, and on Feb. 17, 1944, of burning in the epigastrium. Feb. 17, he was given amphojel, one ounce. Palpation of the abdomen at 8 or 9 P.M. revealed no apparent tenderness, but there was some distention which was thought to be due to the therapeutic pneumoperitoneum. Shortly after this the patient became comatose and he died at midnight.

At autopsy 250 c.c. of dark greenish brown turbid fluid were found in the peritoneal cavity. The parietal and visceral peritoneum showed injection of the vessels and a fibropurulent exudate. There was a perforation of the anterior surface of the first portion of the duodenum. On opening the stomach and duodenum, it was seen that there were two ulcers measuring approximately 0.5 cm. in diameter. One of these was perforated as previously mentioned. The edges of neither of these

TABLE I*

CHART SUMMARIZING THE DATES, TYPE, AND AMOUNT OF FLUID REMOVED, AS WELL AS THE LABORATORY WORK

DATE	AMOUNT OF FLUID FROM RIGHT THORACENTESIS	QUALITY	MICROSCOPIC
1/31	850 c.c.	Creamy pink	Many R.B.C'S
2/1	1350 c.c.	Light pink	
2/2	2700 c.c.	Very light pink	Few R.B.C.
2/3	2100 c.c.	Creamy	No R.B.C.
2/4	2000 c.c.	Creamy	Fat globules
2/5	2440 c.c.	Milky	Fat globules
2/6	2200 c.c.	Milky	Fat globules
2/7	2550 c.c.	Thin milky	Fat globules
2/8	1660 c.c.	Very thin milky	Few fat globules
2/9	1460 c.c.	Cloudy	Few fat globules
2/11	750 c.c.	Straw colored	No fat globules; straw colored
2/12	1100 c.c.	Plasmalike	Plasmalike
2/13	900 c.c.	Plasmalike	Plasmalike
2/14	1500 c.c.	Plasmalike	Plasmalike
2/16	1500 c.c.	Plasmalike	Plasmalike
2/17	1000 c.c.	Plasmalike	Plasmalike
	26,000 c.c.	Total fluid removed from right hemithorax since admission	

LABORATORY WORK

1/29/44—Hg., 85%; R.B.C., 4,900,000; W.B.C., 13,920; Pulse, 78; M., 4; L., 18.
Urine—Sp. Gr., 1.025; Albumin, 0; Sugar, 0; Microscopic, negative.

Blood Chemistry:

	Glucose	CO_2	B.U.N.	Serum Proteins
2/3	87	36%	17.4	5.11
2/7	111		14.3	4.42
2/17		38%	23.6	

Chyle:

2/15	Proteins—2.29
2/17	Proteins—2.0

Kline: negative.

Gastric Analysis (2/7/44):

	Amount	Color	Free HCl	Total Acidity	Lactic Acid
Fasting	120 c.c.	Yellow	10	15	Neg.
Histamine (30 min.)	90 c.c.	Gray	55	81	Neg.

*Table compiled by Dr. Victor Tedesco.

ulcers were raised or undermined. Dissection of the thoracic duct revealed a marked narrowing at a point approximately 3 cm. above the cisterna chyli. This narrowing was caused by fairly dense fibrous tissue surrounding the duct. At the level of the eleventh intercostal space near the vertebral column on the right side there was a small rent in the pleura underneath which the intercostal muscls showed slight hemorrhage. In the right pleural cavity there was 250 c.c. of orange-colored slightly hazy fluid. There was 75 per cent collapse of the right lung and the mediastinal structures were deviated to the left.

Traumatic chylothorax as indicated by the reported results and in our cases is a serious condition. Early recognition of the condition is essential and is not difficult if the condition is considered and if the presence of pinkish-milky or milky fluid is found in a hemithorax.

The chyle should be removed under aseptic precautions, and cultures made. The material may be kept with citrate or heparin as anticoagulants. Administration of the chyle intravenously after determining lack of antigenic property is an important therapeutic measure. Frequent aspirations, culture, and intravenous administration of the chyle are necessary. Fat-free diet reduces the fat content of the chyle, but the amount of fluid is not reduced. The fluid balance must be maintained by parenteral administration which is made difficult because of thrombophlebitis occuring in many of these patients, frequently making it necessary to insert a cannula into the vein after a surgical exposure of it. In an attempt to maintain normal blood protein level, blood and plasma transfusions and amino acids should be administered intravenously. Intravenous fats may prove of value.

No successful direct surgical procedure on the ruptured thoracic duct has been reported. Phrenic nerve crushing has been reported and was used in our case. This procedure is done in a hope that the elevated diaphragm may help occlude the rent in the duct. In our case, pneumoperitoneum was done for the same reason. The prognosis depends upon the closure of the defect in the duct, and the problem is to maintain adequate nutrition until this takes place. An attempt to decrease the amount of chyle formed and procedures as mentioned previously to facilitate occlusion of the open duct are the best methods at the present time.

Unfortunately, in a high percentage of cases, the chyle continues to collect in the hemithorax and, in spite of active therapy, the patient's course is steadily downhill, ending in death. Improvement in the cases which are successful seems to begin with diminution or cessation of the collection of chyle.

Rupture of duodenal ulcer which was the fatal complication in our patient, who was steadily becoming weaker, has not been previously reported. The etiology of ulceration in this 14-year-old boy is not definite. Fat-free diet was started twelve days before he died and he was allowed only water by mouth for the last seven days. This greatly diminished oral intake may have played a part in the etiology of the

ulcer. However, if this is the explanation, we should see duodenal ulceration, and perforation, in children with abdominal conditions requiring limitation of oral intake for equally long periods of time which is generally not the case.

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TOTAL COLECTOMY FOR POLYPOSIS OF THE COLON WITH CARCINOMATOUS DEGENERATION

With a Note on Postoperative Care

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THERE is given herewith the report of a case of diffuse polyposis of the entire colon with carcinomatous degeneration because instances of such widespread disease and of the successful removal of the entire large intestine (colon and rectum) are still relatively uncommon. The removal of such a large segment of the alimentary canal introduces new physicochemical conditions, the management of which, in the light of the modern knowledge regarding protein and other deficiencies and of nutrition in general, has naturally assumed a very important place in those patients who have successfully gone through the total colectomy and who have remained well.

CASE REPORTS

I. P. (Fifth Avenue Hospital No. 1572/42, 6788/42, and 8323/42), a 35-year-old man, consulted his physician for some minor rectal complaint and a small perianal infection was noted. The proctologist who treated the condition discovered a small polypoid mass in the rectum which he removed for biopsy purposes, and a histologic report of carcinoma was returned.

The man was nevertheless later inducted into the Army and the exploratory biopsy procedure was repeated at the Walter Reed Hospital with similar results. He was then discharged from the Army and returned to New York where he was referred to me by his local physician. At this time, and since the disappearance of the local symptomatology related to the perianal infection, there were no local or general symptoms of any kind.

The man was of spare build and considerably undernourished and underweight, but this condition had existed for as long as the patient could remember and was of no special significance to him. The general physical examination was otherwise negative, and the general condition was, nevertheless, relatively good. Locally, nothing could be felt by rectum exclusive of some few polypi which, except for the pathologic report, would not have impressed one as being of any importance.

The removal of the colon was done in three stages: In the first of these, the terminal ileum, cecum, appendix, ascending and about one-third of the transverse colon were removed; the stump of the transverse colon was closed and anchored at the upper angle of the wound subcutaneously; and an ileostomy of the terminal stump of the ileum was established at the lower angle.

There was good reaction from the operation. The wound sloughed moderately, but healing followed progressively though rather slowly. When conditions were thought to be satisfactory, the second stage was done (about six weeks after the performance of the first stage).

At the second stage, the remainder of the transverse colon, the splenic flexure, the descending colon, and most of the sigmoid were removed. The ileostomy was not disturbed. The stump of the sigmoid was closed and again anchored in the lower angle of the wound subcutaneously.

Again there was good reaction from the operation. Healing was slow and the patient was not ready for the third stage for another six weeks.

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The third stage comprised a combined abdomino-perineo-sacral removal of the stump of the sigmoid and the remaining rectosigmoid including the anus. Again there was good reaction from the operation. The healing proceeded as before and the patient was finally discharged from the hospital about six weeks later.

The bulk of the polypi were present in the cecum, ascending colon, and the first half of the transverse colon. In the cecum and proximal ascending colon, the polypoid excrescences formed a thick layer. Further onward, the abundance and thickness of the polypoid layer began to diminish. In the left half of the colon, there was a sparse distribution. In the rectosigmoid, the lesions were relatively few in number. It was very fortunate that the nature of the condition was discovered from the few rectal lesions available for biopsy and study. The distribution of the polypi could be recognized in the roentgenologic studies taken before operation.

The pathologic report on the polypi in the right colon was as follows: Diffuse polypoid adenomatosis showing extensive precancerous changes and chronic inflammation with focal hemorrhage. Pericolonic lymph nodes showed lymphocytic and endothelial hyperplasia. The appendix showed atrophy, fibrosis, and obliteration. (Spear.)

DISCUSSION

Polyps occur in the lower bowel in one-third of the patients studied proctosigmoidoscopically, while roentgenographically polyps occur in the entire colon in one-half of the patients studied.

Polyps are related to the lymphadenoid accumulation in the alimentary tract. Mechanistically they seem to interfere with the proliferative changes in the neighboring epithelial and glandular cells and produce follicle and cystic formations which become projected into the lumen of the bowel as polypoid structures. The development of either sessile or pedunculated bases is dependent upon mechanical factors (muscular contraction, etc.), on any connective tissue replacement at the base or in the core of the polyp, and on the extent of the original lymphadenoid accumulation. These structures form the "benign adenomas" and the latter occur much more frequently than is usually thought. The formation of these structures in the alimentary tract requires a hereditary disposition possibly, but, much more important, there is also present a continuous stimulation of the tissues due to some chronic irritation. Nevertheless, the importance of either one or both of these factors varies in individual cases.

There apparently are two factors that determine transformation of a benign polyp into a malignant one: (1) the degree of undeterminable individual tendency either inherited or acquired toward such a development; and (2) the degree, intensity, and duration of repeated chemical and mechanical irritation especially prone to occur in the large intestine. In the presence of primary carcinoma of the colon or rectum, polyps are commonly found in addition to the primary lesion (about 33 per cent)

and in many of these malignant degeneration can be recognized. Such changes may account for many if not all of the recurrences in the intestinal cases.

In one of my patients, three individual tumors developed in the sigmoid, the rectum, and at the ileocolic junction during a period of three years. Each had all the earmarks of an independent primary tumor and each was treated as such. Twenty years after the performance of the last resection, the man is alive and well and going about his usual business. It is undoubtedly so, that these developed upon the basis of individual adenomatous polypoid structures which independently had undergone malignant degeneration, probably because of similar general (inherited?) and/or local irritative causes.

Factors in the Successful Treatment of Colonic Polyposis.—A number of immediate and late problems are present and must be met in the correct and successful handling of this type of lesion and of an operation of such magnitude.

1. Preoperative preparation. This comprises two parts: (1) thorough cleansing of the large bowel by adequate catharsis in colonic irrigations; and (2) the building up of the general condition of the patient to the highest possible level.

Building up of the general condition of the patient includes (1) the rectification of any cellular and hemoglobin deficiency of the blood to at lease 85 per cent of the normal; (2) rectification of any protein deficiency in both blood or tissue not only to the normal but, if possible, to an excess; (3) rectification of any abnormal hepatic function with which both Items 1 and 2 are intimately bound up. Any other associated condition, particularly cardiorenal vascular disease, should similarly receive appropriate and adequate care. (See Section 5).

All of this preparation is necessary because all of these patients are in relatively bad condition when they come to operation; they are underweight and undernourished; they show important grades of anemia; laboratory studies show protein deficiencies; and the cardiorenal vascular apparatus frequently shows impairment.

2. Unless there is absolute contraindication, spinal anesthesia is the method of choice.

3. Stage operations. Resection of the entire colon must never be done in one stage. This would be much too great a procedure for anyone—especially those with this disease, in which all the safety factors, usually present in other types of operatively treated disease, are dissipated to their lowest ebb. The colectomy should be done in three stages as this gives the greatest safety factor and the greatest chance for success.

4. Infection. An extensive lesion of this kind facilitates the presence within the bowel of more than the usual amount of infectious material and adds to the danger of the intestinal resection. This can be avoided or controlled by careful and thorough bowel cleansing preoperatively, by meticulous attention to one's operative technique and postoperative

care, and by the judicious use of chemotherapy. In regard to the latter, one should be very careful with the local intraperitoneal use of the sulfonamide drugs. When used in too large amount, they tend to cake. Thereafter they act as foreign bodies; abscesses then form which may be difficult or impossible to contend with and I know of one case in which a fatality occurred because of this reason.

5. Postoperative care. In reality this is a continuation of the preoperative building up of the general condition of the patient. It is to be presumed that the rectification of blood, protein, or hepatic deficiencies to somewhere near a normal status had been carried out to a fairly satisfactory state before operation. Operation, nevertheless, will tear down some of this good work and the patient will revert, as far as these factors go, to something like the previous condition. According to the newer knowledge, this reversion is due to the largest part to a destructive and/or inhibition alteration of the protein function following an injury, in this case an extensive operation. This is what makes the factor of nutrition and the protein metabolism the crucial factors for the recovery of the patient and his subsequent good health.

We must then begin all over again and rebuild the lost ground using every means at our command* and including: (1) an enriched protein diet; (2) intravenous alimentation with glucose, salt, minerals, vitamins, and aminoids; and (3) transfusions of plasma and/or whole blood. All of this must make good to an excess the abnormally large losses which are bound to take place in the unutilized discharge from an ileostomy. The criteria, upon which one measures and decides the success of our efforts, are based (1) upon the visible state of nutrition, (2) upon the gain in weight, (3) upon the character of the healing in the wound, (4) upon liver functional tests, the state of the hemoglobin and cellular content of the blood, the state of the proteinemia, and, finally, (5) upon the general condition of the patient.

If success is to follow our postoperative plan of care, all of these items and factors must be kept at as high a state of efficiency as possible. The basis of success is adequate nutrition and a satisfactory protein metabolic effort. All of the other factors necessarily depend upon this. Any protein deficiency, therefore, needs immediate attention.

In accordance with the degree of the deficiency, the length of time it has existed, the nature of the operation, etc., the protein deficiency should be replaced as in the preoperative preparation, of which the postoperative therapy is only a continuation, with all permissible intensification of the program.

Immediately after operation, it will be necessary to begin with parenteral methods. Then one proceeds as soon as convenient or practical to a combination of parenteral and enteral methods, and finally, as soon as the patient's digestive powers are fully available to the necessary capacity, to enteral methods alone.

*Wilensky, A. O.: The Treatment of Protein Deficiencies, Surg. Gynec. & Obst. (In Press.)

Immediately after operation, parenteral methods of nutrition include:

(1) the use of plasma in sufficient amounts, combined with (2) transfusions of whole blood. A combination of the two seems to answer the purpose better than either one alone. In addition, the fluid balance is kept up by glucose and saline infusions, to which solu-B (vitamin B) can be advantageously added. In accordance with the excess loss through the ileostomy, large quantities of fluid are needed, up to 4,000 to 5,000 c.c. daily, but the danger of overloading the vascular apparatus should be kept in mind in these otherwise debilitated patients. I have formed the habit of adding to each 1,000 c.c. of infusion 30 Gm. of aminoids (protein hydrolysate) and have found this of great use.

As soon as possible, oral feeding is instituted and increased as follows:

1. The dietary protein intake is increased to 150 to 180 Gm. per day. Usually the patient will not or cannot take more by mouth.

2. Gastric intubation is used, if possible. This may add as much as 50 per cent. But if too much is given, distention results, the patients develop diarrhea, and much protein is lost.

If, owing to digestive or other disability, the amount of protein is not sufficient, the discrepancy should be made good by adding a sufficient quantity of amino acids dissolved in, or mixed with, the food. This is easily done in the mouth feeding, and even more easily accomplished by intubation.

In the beginning, it is usually necessary to bolster the oral feeding with parenteral alimentation either by plasma or by whole blood, and this should not be avoided until the oral method is firmly established and is sufficient.

Later and after the patient leaves the hospital, excess and enriched protein nutrition should be continued for the reason that the ileostomy continues to discharge abundantly, and much is lost before complete utilization. Occasionally, even at this later period, bolstering up with parenteral methods is necessary—plasma, blood, and transudate fluids (ascitic or pleural). The last is a valuable method and decreases the expense.

At all stages of the postoperative care, alimentation, either oral or parenteral, must not exceed the toleration of the individual patient, otherwise the purpose would be defeated, much harm would be done, and convalesence from the operation would be interrupted.

At all stages of treatment, protein hydrolysates in the form of aminoids form a valuable addition to oral or parenteral alimentation. In the early stages, it can be used parenterally by adding the aminoids to the infusions as indicated previously. Later, aminoids can be added to the diet to enrich the protein stores. In long-standing cases of undernutrition, the oral use of these preparations finds an important and chief place.

Review of Recent Meetings

FIFTY-SIXTH ANNUAL MEETING OF SOUTHERN SURGICAL ASSOCIATION

GEORGE D. LILLY, M.D., MIAMI, FLA.

THE fifty-sixth annual session of the Southern Surgical Association was held at the Homestead Hotel, Hot Springs, Va., Dec. 5, 6, and 7, 1944. **Alton Ochsner** presided and **Alfred Blalock** served as secretary. **I. A. Bigger** was chairman of the Committee on Arrangements.

Colonel Arnold Griswold began the scientific program by presenting a discussion of **Unusual Lesions of the Stomach.** Several cases were presented in which gastric tumors were encountered in the proximal portion of the stomach. The advantage of transthoracic approach to such lesions was demonstrated. It was pointed out that an approach through the left tenth rib bed, with splitting of the diaphragm and crushing of the phrenic nerve, offered excellent exposure of this surgically inaccessible region. Infection was not encountered frequently, and the additional time consumed seemed to be worth while because of the better exposure.

William F. Rienhoff analyzed **The Results of the Surgical Treatment of Peptic Ulcer of the Duodenum in a Series of 260 Cases.** Intractable pain, repeated hemorrhage, and vomiting were conditions which made surgery desirable. A Polya-Balfour type of resection was employed, with removal of one-third of the stomach and an anticolic anastomosis. He reported that 78 per cent of the patients in this series were entirely well, 12 per cent improved, 8 per cent failed to improve, and 2 per cent died; 10 per cent had postoperative hemorrhage and 8 per cent developed marginal ulcer.

Samuel F. Marshall presented **A Plan for the Management of Gastrojejunocolic Fistula.** In reviewing the literature on this subject, mortality rates ranging from 25 to 63 per cent were encountered. Most deaths were attributed to shock, hemorrhage, and peritonitis. It was observed that most of the fourteen cases studied developed following posterior gastroenterostomy. First, a jejunal ulcer developed, and then this involved the colon. The greatest mortality was found to result from a single disconnecting operation. Dr. Marshall believes it is safer to do a radical two-stage operation; first, the terminal ileum is anastomosed to the descending colon, and second, the ascending and transverse colon are resected. In one case the second stage was not necessary. There was one death in this series of fourteen cases.

Lieutenant Colonel Harry J. Warthen outlined **A Plan for Handling Emergency Surgery in Army Air Force Hospitals.** Rooms were set up with full equipment and helpful charts, which enabled those not too experienced in this type of work to refresh their memory. Dr. Warthen pointed out that such a plan might be applicable to civilian emergencies and could be adapted to civilian hospitals.

Lieutenant Colonel Alfred R. Shands, Jr., and **Major Randolph L. Clark** discussed **Surgical Problems in Aviation Medicine.** The anatomic damage caused by aircraft accidents was analyzed. It was found that the sudden deceleration as-

Received for publication, Feb. 23, 1945.

sociated with such accidents was the most frequent cause of death and bodily injury. It was pointed out that such deceleration forces could be tolerated by the human body with less damage if they were applied transversely to the long axis of the body. Gunfire was found to be a frequent cause of injury. The quick freezing of tissue caused by exposure at high altitudes was discussed. The best treatment for this is believed to be procaine block of the sympathetic nerve supply of the involved extremity, followed by pressure dressings and inhalation of oxygen. Parachute injuries were considered. The importance of a "free fall" was stressed. Flyers, when jumping from high speed planes, should delay opening their parachutes until they decelerate to the 200 m.p.h. speed of a free falling body. The frequency of injury when parachuting from combat planes was studied. Thirty-seven per cent of all combat parachuters were injured; one out of eight was killed. Many jumped at too low an altitude, and 20 per cent hit their aircraft when bailing out.

Colonel B. N. Carter and **Lieutenant Colonel Michael E. DeBakey** discussed **Current Consideration of War Surgery.** The great experiences in traumatic surgery afforded by war casualties were reviewed. Shock is still a matter of most importance. Plasma has been of value on the battlefield, but the general treatment of shock is still of great importance, and, if whole blood is available, it is much better than plasma in these cases. The wound management of war wounds is not comparable to that in civilian practice because of battle condition. Secondary closure is now the general practice in military surgery. It has been found that secondary suture has been successful in from 75 to 90 per cent. The use of sulfonamide drugs has been most disappointing; their use may prevent death from sepsis, but they do not aid in primary healing. Penicillin has been found to be much better than sulfonamide drugs, but its chief value lies in the fact that it makes good surgery possible at an earlier period in infected cases. Mechanical cleansing is still the most important factor in preventing infection.

Lieutenant Colonel Brian Blades discussed the use of **Penicillin as an Adjunct to the Surgical Treatment of Acute and Chronic Empyemas.** He described his experience in treating empyema by injecting 50,000 units of penicillin into the empyema cavity on alternate days, and came to the conclusion that it was of little, if any, value. Surgical drainage was done in eleven of the thirteen cases. He felt that penicillin was of no value as an irrigating agent, but he believed it might be of value as a preoperative measure in surgery of chronic empyema. It is his opinion that systemic penicillin might be of value in preparing patients, suffering from suppurative lung disease, for surgery. His conclusion was that empyema is an abscess, and requires surgical drainage.

Arthur H. Blakemore described **A Nonsuture Method of Blood Vessel Anastomosis** and reported the progress which he has made with the use of vitallium tubing lined with free vein grafts. His most interesting work has been in perfecting a portal-caval anastomosis in which the splenic vein was anastomosed to the left renal vein. He found that such a venous anastomosis relieved portal hypertension and relieved those patients who were suffering from ascites.

Loyal Davis reviewed **The Surgical Principles Underlying the Use of Grafts in the Repair of Peripheral Nerve Injuries.** Dr. Davis pointed out that nerve injuries should be recognized at an early period, and the separated nerve ends should be brought together as soon as possible. The practical management of nerve injuries under battle conditions was discussed. The use of fresh autogenous, or homogenous, grafts of nerves of the same size was described. Such grafts are far from satisfactory and should be used only as a last resort. Considerable difficulty has been encountered in connection with fibrosis at the level of the distal suture. Hope was expressed that plasma sutures may help.

George H. Bunch discussed **Mucoid Disease of the Appendix.** Dr. Bunch described the clinical picture associated with this disease, reviewed the statistics, and considered the complications. He reported on four of his own patients, two with mucoid disease of the appendix, and two with mucoid cysts of the ovary.

Thomas B. Aycock and **Edward M. Farris** reported **The Effects of Sulfonamides on the Mortality Rate in Acute Appendicitis.** They analyzed the results in 1,151 cases. Approximately 600 patients were operated upon prior to June, 1940, with a mortality rate of 5.2 per cent. After that date, 500 were operated upon with a mortality of 1.2 per cent. Credit for this improvement in mortality was given to sulfonamide drugs which were used routinely in the latter group.

Major Barnes Woodhall and **Lieutenant Colonel R. Glen Spurling** discussed the use of **Tantalum Cranioplasty for War Wounds of the Skull.** They felt that tantalum is the best available material for cranioplasty. They found it to be inert when buried in tissue. It is easily shaped and is applicable to fresh wounds.

J. Albert Key created considerable controversy by contending that **Disc Lesions Are the Chief Cause of Low Back Pain.** Dr. Key believes that 90 per cent of all low back pain is due to disc pathology. He feels that those patients who have been reported as having persistent pain after a disc operation may be placed in one of the following categories: (1) incomplete removal of the ruptured disc; (2) removal of the wrong disc; (3) development of another ruptured disc; (4) adhesions; (5) arachnoiditis; (6) undiagnosed tumor. Dr. Key feels that adequate removal of the diseased disc is all that is necessary. He has abandoned spinal fusion in connection with this operation. This paper was discussed by **George Bennett, Paul Mogunson, Guy Caldwell, G. B. Rhodes, W. J. Mixter,** and many others, all of whom disagreed with Dr. Key.

George M. Curtis discussed **The Surgical Significance of the Accessory Spleen.** He stressed the importance of looking for accessory spleens when splenectomy is done, and pointed out that many patients who are not benefited by splenectomy fail to respond because accessory spleens are overlooked. The incidence and location of accessory spleens were reviewed.

Roy D. McClure and **Conrad R. Lam** reported their **End Results in the Treatment of Hyperparathyroidism.** Six cases were presented, and the importance of associating hyperparathyroidism with the clinical picture of Recklinghausen's disease, osteoporosis, and renal lithiasis was stressed. A case of fatal hypoparathyroidism which developed following the removal of a single parathyroid tumor was reported.

Charles C. Green discussed **Desmoid Adenoma** and reported on cases of his own.

G. V. Brindley presented a paper on **Sacral and Presacral Tumors.** He reported on cases of his own and described the clinical characteristics of presacral dermoids, chordomas and ependymal cell tumors. A plea was made for adequate exposure and radical resection with a cautery knife.

Lieutenant Colonel Lloyd G. Lewis presented four **Cases of Severe Injury to the Pelvis** and considered the problems involved in this type of injury. He pointed out that pelvic injuries were frequently complicated by injury to the urethra, bladder, and ureters, and stressed the importance of adequate drainage and a long delay in repair until infection subsides.

Harold L. Foss considered the advantage of **Pan Hysterectomy** in certain benign lesions as well as in carcinoma, and concluded that total hysterectomy is a more dangerous procedure and should be used only when a diseased cervix is present. He pointed out that stump carcinomas occur in from 1½ to 2 per cent of cases,

and stated that this is less risk than the increased mortality associated with total hysterectomy, as compared with a subtotal hysterectomy. He described his own technique in which he uses a right angle clamp.

Leo Brady described a **Method of Constructing a Vagina** in which he used the labia minor to construct pedicle flaps with which to line the artificial vagina. He reported one case in which what seemed to be a uterus turned out to be a pelvic kidney. He called attention to the fact that congenital abnormalities of the urinary system are frequently associated with abnormalities of the genitals. He suggested that an intravenous pyelogram be done in all such cases.

Lawrence R. Wharton reported a case of **Spontaneous Perforation of the Rectovaginal Septum Following Construction of the Vagina.** Five and one-half weeks after an easy operation to create an artificial vagina the balsa wood vaginal form perforated the rectovaginal septum. It was pointed out that a long form should be used, so that the perineal muscles could not force it through the rectum during coughing or straining at stool.

Kenneth L. Pickrell and **Richard K. Richards** described **A Method of Shortening the Recovery Period Following Pentothal Anesthesia.** They conducted a clinical and experimental study of the antagonistic effect of metrazol in patients and animals under sodium pentothal anesthesia, and concluded that the use of metrazol for this purpose was of definite value in shortening the recovery period after the use of this type of anesthesia.

Warfield M. Firor and **George O. Gey** reported their work with malignant cell cultures. After they carried on continuous cultures of fibroblasts for many months, some alterations began to appear in certain cells. When these altered cells were inoculated into rats, four different types of sarcoma developed. By observing these cells during the early stages of their change, it was determined that the malignant cells put out a substance which digested or altered the cohesive intercellular substance and caused the cells to lose their usual arrangement. A fascinating microscopic motion picture was shown which actually demonstrated this process.

J. M. T. Finney, Jr., reported his experience with **Primary Carcinoma of the Gall Bladder.** He feels that cholelithiasis and chronic cholecystitis may lead to the development of carcinoma of the gall bladder, and on this basis he made a strong plea for early operation upon diseased gall bladders. He reported eleven of his own cases, and concluded that once carcinoma of the gall bladder is well established the prognosis is hopeless.

Arthur W. Allen presented **A Method of Reconstruction of the Bile Ducts.** He reviewed the causes of surgical injury to the bile ducts, the symptoms of such injury, and the principles of repair. He described the use of an isolated loop of jejunum from which the fecal stream had been diverted as a site for the transplantation of the remaining portion of the common or hepatic ducts.

Frederick A. Coller summarized his experience with the **Treatment of Carcinoma of the Colon** during the past five years. In his large series 83 per cent were found to be resectable, 64 per cent had a chance of a cure. His operative mortality was 4 per cent, and his over-all mortality was 7½ per cent. He did not use sulfonamide drugs in the peritoneal cavity or by mouth at the time of surgery, and neither sulfaguanidine or sulfasuxidine was employed as a preoperative measure. These drugs were avoided in order to establish a control group for later comparison with a group in which the drugs will be used.

Everett I. Evans discussed his experience with **The Treatment of Clinical Shock With Gelatin.** He found that when especially prepared gelatin is given

intravenously the entire amount remains in the blood stream for at least eight hours, and this solution has proved to be a very satisfactory blood substitute. This preparation will soon be available commercially, and will make a satisfactory blood substitute in localities where whole blood is not available.

Francis M. Massie described **The Use of Heparin in the Abdomen** for the prevention of the development of intraperitoneal adhesions. He reported that this solution did prevent the development of postoperative adhesions, but he found it to be a very dangerous drug. Its use was complicated by wound bleeding, failure of wound healing, ileus, and hemorrhage. In spite of these objectionable features, he feels that its use is justified in a few carefully selected cases.

Lieutenant Colonel T. C. Thompson described a procedure for **Quadricepsplasty.** He found that in some thigh injuries, scaring and fibrosis of the quadriceps femoris muscle interfered with function. In such cases he removed the injured vastus and excised the scar tissue from the remaining muscles to improve their contractility.

John E. Cannaday reported on his **Additional Experience With the Use of the Cutis Graft in Surgery.** He has found that cutis grafts are more satisfactory than fascia in the repairing of hernias, because fascia is strong in only one direction. He reported his excellent results in 105 cases, and described the technique which he employs.

G. B. Hodge, Keith S. Grimson, and **H. M. Schiebel** reported **The Surgical Results of a Revival of Radical One-Stage Excision and Stripping of Varicose Veins Coupled With Pressure Bandages and Early Activity.** They reported that 195 patients with large varicosities had a radical stripping operation done; 102 of these were followed for ten to twenty-seven months, and they felt that very good results were being attained.

Book Reviews

Gynecology and Gynecologic Nursing. By Norman F. Miller, M.D., and Virginia Bryant, R. N. Philadelphia, 1944, W. B. Saunders Company.

This new nurse's textbook will be widely welcomed. The authors have wisely used the physiologic approach in discussing diseases of the female generative tract. In addition, nursing techniques are clearly presented.

The handling of the subject matter is accurate, concise, and conservative, and for the most part complete. The importance of nursing care in the prevention of postoperative thrombophlebitis and embolism might be given additional emphasis. Some discussion of local anesthesia in gynecology would also be a desirable addition.

Body mechanics are discussed in Chapter 8. Some might think discussion of such a topic foreign to gynecology, but the authors have shown clearly the importance of this subject often neglected by orthopedists as well as gynecologists.

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By **MAURICE FELDMAN, M.D.,** Assistant Professor of Gastro-Enterology, University of Maryland; Associate Roentgenologist, Sinai Hospital; Assistant in Gastro-Enterology, Mercy Hospital, Baltimore. Second edition, 1945, 778 pages, 551 illustrations, $7.00.

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"This book covers completely the whole subject of roentgenology of the gastrointestinal tract. It represents a tremendous amount of work on the part of the author. Each disease described has its clinical side presented sufficiently to allow both the clinician and the roentgenologist to obtain a fairly complete picture of the whole subject. The book is well written, has a large index, and can be recommended for the use of clinicians and roentgenologists." *Journal A. M. A.*

"Throughout the volume Dr. Feldman discusses not only the roentgenological considerations of disease of the gastro-intestinal tract but also the clinical, surgical, and pathological aspects. The book will be appreciated particularly by roentgenologists and gastro-enterologists but is recommended to any student or physician who wishes to learn more about the diagnostic roentgenology of the digestive tract. The book is well written, is characterized by its completeness, and is recommended without reservation." *Surgery, Gynecology and Obstetrics.*

"This book is crammed with valuable information. It is the only volume of its kind in all English medical literature, representing a lifetime of work and study into the many problems confronting us in application of the X-ray to clinical investigation of the digestive tract. The material is well written and the illustrations excellent. No library of individual, school, college or hospital can be considered to be complete without a copy of this book on its shelves." *New York State Journal of Medicine.*

"It is a book essential alike for the roentgenologist and gastro-enterologist, as well as to the internist who, not satisfied merely to accept the reports of others, wishes to satisfy himself about the interpretation of a series of gastro-intestinal films. A diligent study of this book will serve as an excellent preparation for one who intends to specialize in this field." *Review of Gastro-enterology.*

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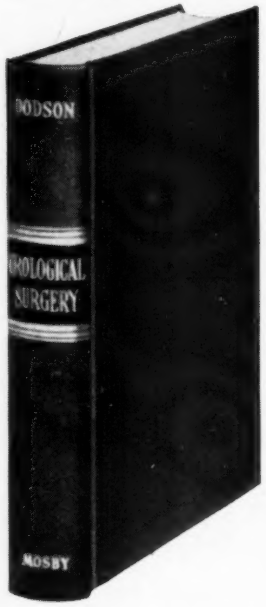

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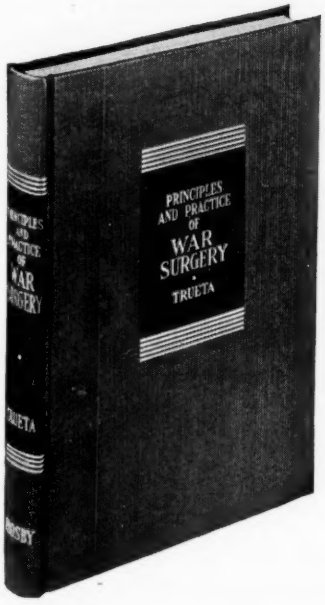

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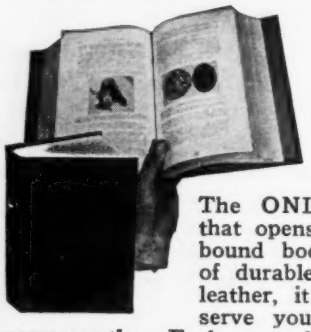

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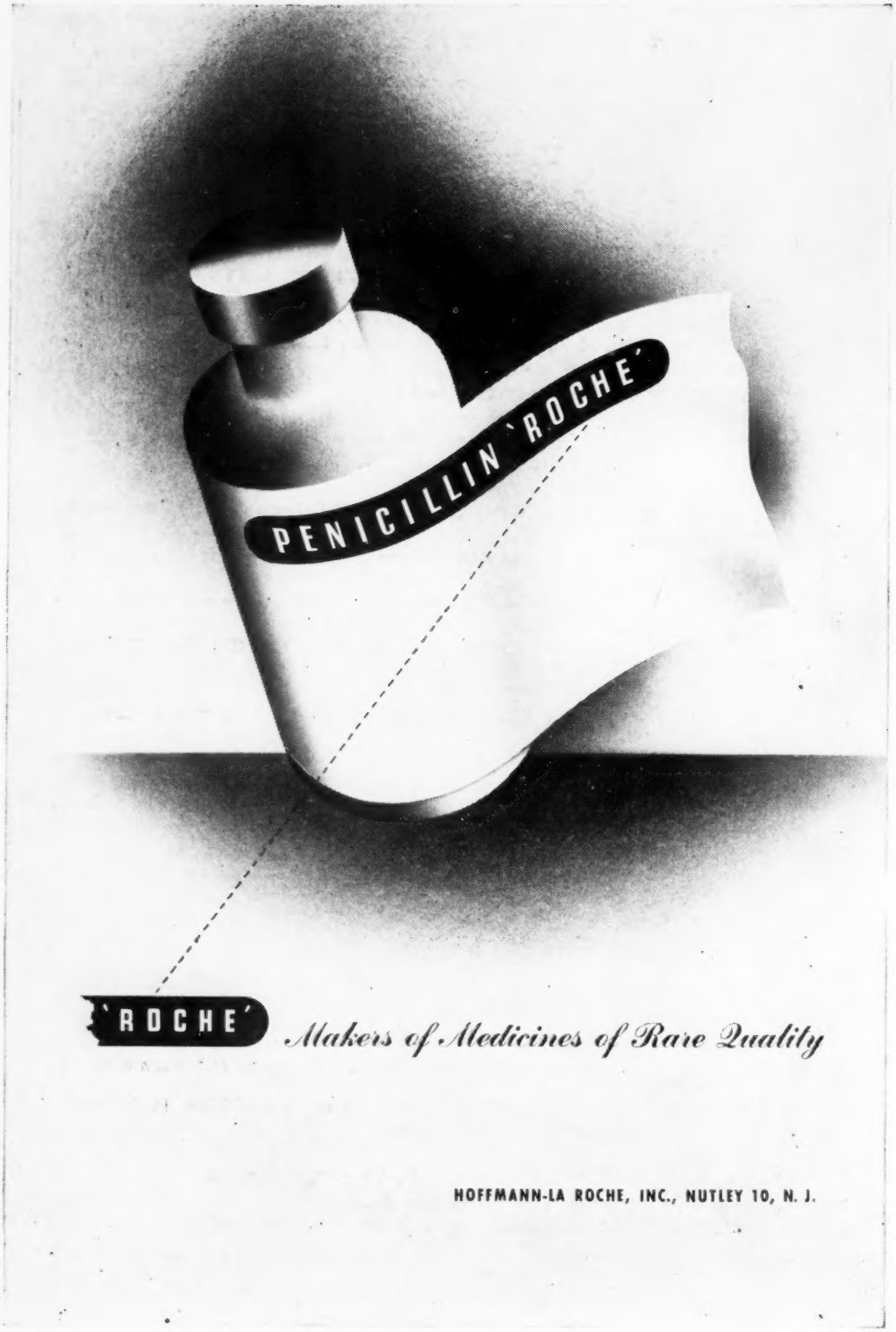
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